ROYAL COMMISSION ON MATTERS OF HEALTH AND SAFETY

ARISING FROM THE USE OF ASBESTOS IN ONTARIO

CHAIRMAN: J. STEFAN DUPRE, Ph.D.

COMMISSIONERS: J. FRASER MUSTARD, M.D.

ROBERT UFFEN, Ph.D., P.Eng., F.R.S.C.

COUNSEL: JOHN I. LASKIN, LL.B.

APPEARANCES: P. Casgrain, Quebec Asbestos Mining Association

J. McNamee, Government of Ontario

L. Jolley, Ontario Federation of Labour

T. Hardy, Asbestos Information Association of North America

180 Dundas Street
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VOLUME XXXII

ROYAL COMMISSION ON MATTERS OF HEALTH AND SAFETY

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THE FURTHER PROCEEDINGS OF THIS INQUIRY RESUMED PURSUANT TO ADJOURNMENT

APPEARANCES AS HERETOFORE NOTED

DR. DUPRE: I see we have a full contingent from summer school back for the fall term.

Are you ready to proceed, counsel?

MR. LASKIN: I am, Mr. Chairman. I have Dr. John Dement with us.

DR. DUPRE: May I please, on behalf of all of us, Dr. Dement, greet you most warmly. You became something of a household word during our summer school, and we are very happy to have our professor in the flesh today.

Miss Kahn, would you swear in the witness, please?

DR. JOHN DEMENT, SWORN

EXAMINATION-IN-CHIEF BY MR. LASKIN

Q. Dr. Dement, you have in front of you a brief of some of your publications, and for our purposes we have given it an exhibit number and it's exhibit forty-three, so that if we refer to any of the articles in there we will refer to them by their tab numbers and the exhibit.

Q. (cont'd.) Because there is not a curriculum vitae in exhibit forty-three, could I just briefly go over with you your educational background and employment?

I take it you had a bachelor of science in chemical engineering from North Carolina State?

A. That's right.

Q. Then a master of science in environmental health and industrial hygiene from Harvard University School of Public Health?

A. That's correct.

Q. Then your Ph.D. from the University of North Carolina?

A. Right.

Q. What is your present occupation?

A. My present occupation, the job is titled Health and Safety Manager with the National Institute of Environmental Health Sciences in Research Triangle Park, North Carolina.

Q. I take it that's a fairly recent appointment?

A. Before that, Deputy Director of the Division of Respiratory Diseases Studies for NIOSH, Morgantown, West Virgina.

Q. All right.

MR. LASKIN: Let me file, and I'll make a copy available, your complete curriculum vitae, and perhaps we can just mark it as tab twelve of exhibit forty-three.

EXHIBIT #43, TAB #12: The abovementioned document was then produced and marked.

MR. LASKIN: Q. All right. I take it you are going to give us a brief overview, in any event, of your major recent publication which is at tab nine, so by all means, Dr. Dement, the room is yours.

A. From discussions that I have had with members of the committee, and correspondence from Linda, I take it that the paper that was presented at the Fifth Inhaled Particles Conference in Cardiff in September of 1980, has been the subject of...at least the subject of discussion in this committee, so I thought what I might do is just briefly review the study itself, and the findings and sort of my interpretation of what the findings mean. Then we can field questions.

This committee has, of course, reviewed a voluminous amount of data of asbestos and the health consequences of the least occupational exposures.

Suffice it to say that the majority of epidemiologic studies that have been conducted among worker populations have involved populations exposed to a mixture of different types of asbestos fiber, this being the nature of most industrial operations. The mixtures are used because of the requirements of the product being made.

The plant that I chose to study was a rather unique plant in that only chrysotile had been used, except for one brief period of time when crocidolite yarn was imported, about two thousand pounds in the history of the plant, and rather recently.

But chrysotile only. The source of the chrysotile came from both Canada and Rhodesia, and there were overlapping periods where these different fiber types were used. So it's unique in the fact that chrysotile could be studied.

Several other important characteristics of the plant: It was a stable, a relatively stable population. It was also a very old plant - in fact, one of the oldest in the United States, and the first published information concerning the association between asbestos exposure and respiratory cancer actually came from this plant, posed by Lynch and Smith in the

THE WITNESS: (cont'd.) autopsy case which revealed cancer in addition to asbestosis. So it has some historical prospectus.

Additionally, the plant was pretty unique in that it was pretty progressive in the application of engineering controls, even back into the late thirties, and in fact the U.S. Public Health Service studied this plant in conjunction with their 1938 studies of asbestos plants in North Carolina, and used this plant as a demonstration plant of engineering controls...I would say state of the art engineering controls for an asbestos textile plant at that time.

So it was pretty unique and by about 1940, most controls in that plant were in place. In fact, these remained pretty stable throughout the study period that I studied in 1940 to 1975.

Lastly, but probably most important, there was a good quantity of environmental data historically in this plant that could be used to estimate exposures.

I chose to study actually, I guess, two sources - the Ph.D. dissertation which you have is a study of both black and white males. I have only reported in the literature on the white males. The reason for doing that is the black male population in the dissertation, I didn't feel, has been followed up completely enough to warrant publication at this time, so I report on the white male data which is the subject of the Cardiff paper.

The cohort consisted of seven hundred and sixty-eight white males employed more than six months in asbestos textile operations, between 1940 and 1975...between 1940 and 1965, the entry date. The cohort was followed through 1975.

I used a number of different sources of information to ascertain cohort vital status as of the cutoff study date, and we were successful at the time of the Cardiff paper

THE WITNESS: (cont'd.) in finding a good portion of them. Of the seven hundred and sixty-eight males, there were a hundred and ninety-one who were known to be deceased, there were twenty-two for which we had no vital status information. With those twenty-two we are assuming that they were alive and they were counted as alive to the end of the study date in 1975, thereby we made a conservative estimate that their person-years of observation have contributed to expected deaths.

I might add since this 1980 study we have completed further followup and this number is less than half the vital status so far and it hasn't changed the results of the mortality study.

Overall, just to back up a bit, for the epidemiologic investigation we took into account age, race, sex, calendar year and cause-specific mortality in calculating the expected deaths. The standard population used to calculate expected deaths was that of the white male population of the U.S.

In this group we observed a hundred and ninety-one deaths through the end of the study period 1975, and expected a hundred and forty-one. So the SMR, the ratio of observed to expected, was about a hundred and thirty-five...an SMR of one thirty-five.

That was significantly increased.

When you look at specific causes, we found that there are really only two causes that were significantly in excess and that was malignant neoplasms of the lung, trachea, bronchus and the lung, or ICDA rubrics 162 and 163, and nonmalignant respiratory diseases. Of that, the group 'other respiratory diseases', which included the pneumoconioses, there were eighteen observed and slightly over three expected.

So this is pretty consistent with findings of other asbestos cohorts.

THE WITNESS: (cont'd.) When you looked at this group further by duration since first employment, we found that in fact before fifteen years since initial employment there were no lung cancer deaths...in fact, a deficit of lung cancers...which increased rather markedly to the greater-than-thirty groups.

So this is, again, very consistent with an occupational etiology.

We attempted, using the industrial hygiene data available, to reconstruct exposure estimates between 1930 and 1975, and for this study we had about fifty-nine hundred environmental samples which we collected from a number of different sources.

One of the things that we had to do was make a conversion. Many of the early samples prior to about 1965 were collected by the impinger method recommended by the U.S. Public Health Service.

Between 1965 and about 1971, there was a period of overlap where impinger samples and membrane filter samples were collected.

After about 1971, the membrane filter method was exclusively used.

In order to arrive at the exposure estimates, the first task was to estimate some conversion between the two sampling methods to get into some common measure. Fortunately, there were two sources of data that were pretty unique and useful for this purpose.

The first was the study conducted, actually, by the U.S. Public Health Service in 1965, when a hundred and twenty paired, side-by-side samples by the two methods were collected. The object of the study was actually this, to compare the two methods.

Second of all, between 1968 and 1971, both the impinger and the membrane filter samples were used to sample

THE WITNESS: (cont'd.) in various textile operations, so this provided two independent data sets for estimation of that conversion.

I won't go through all the details of process, but basically we found that in all textile operations besides preparation, a conversion of three fibers per c.c. per one million particles per cubic foot was appropriate, and textile, a larger conversion was indicated by both the independent sample sets of independent data, and for that we used eight.

That's probably an overestimate of that actual conversion, but we used it realizing it was perhaps a bit high.

Anyway, using those conversions we converted all of the data to impinger concentrations. Using the industrial hygiene data, we also tried the model plan operations, taking into account jobs, operations and periods of time and changes in engineering controls. We conducted a detailed survey of all changes in plant processes and controls over the study date, so that we knew when controls and process changes were put in.

So we arrived at an average exposure for each job and textile over the study period. That was combined with detailed work histories from each individual's employment record to caclulate the accumulative exposure, product of exposure level times his time of exposure, throughout the study period.

So we combined these data to look at dose response for specifically lung cancer and nonmalignant respiratory disease.

The data that we generated from a dose-response analysis, for lung cancer we found a linear, or at least adequately described as linear relationship between dose, cumulative exposure and lung cancer SMR. That is, in the lowest exposure category we found eight lung cancers observed versus three point five nine expected, for slightly over a twofold excess. In our next-to-highest dose category we found nine lung cancers versus less than one expected,

THE WITNESS: (cont'd.) for an SMR of about nine seventy-eight.

For nonmalignant respiratory diseases, we found again, although not as linear as the lung cancer trend with dose, perhaps adequately described as linear based on the data that we had, but again, a very marked dose-response relationship.

Whereas, in the next-to-highest dose category we had seven other nonmalignant respiratory disease deaths observed versus zero point three eight expected.

In this group we found only one mesothelioma. I think that's fairly significant and pretty consistent with other studies of chrysotile-only exposure.

I might add that it is probably as likely that this is under-diagnosed in most asbestos cohorts.

We attempted to gather as much pathology data as we could. In fact, a local teaching hospital in the area served as a reference laboratory in pathology and had maintained a pretty good record set, and these data were all reviewed in attempts to find additional cases, and we found none.

Just a couple of comments on other risk factors for this group is appropriate. We chose to use the U.S. white male population as the standard reference population for calculation of expected deaths. In fact, when you look at age-adjusted death rates for the state in which the plant was located, with the U.S., they are very much identical.

The county in which the plant was located has a significantly elevated lung cancer death rate for the period 1950 to 1969. Two comments are appropriate, I suppose. First of all, this county is the site of a large shipyard which operated back before World War Two, with peak involvement as high as roughly twenty-nine thousand persons.

There were two studies conducted by our National

THE WITNESS: (cont'd.) Cancer Institute which demonstrated a strong association between shipyard employment and elevated lung cancer within a county.

If you look at contiguous counties, you find that these fall more back into line with the U.S. death rates.

Another risk factor for lung cancer is smoking. Fortunately, we had some data to look at the prevalence of smoking in this population and compare it with our reference population. This came from U.S. Public Health Service questionnaire data collected in 1964, and again in 1971, using roughly the BMRC respiratory questionnaire.

We found that asbestos workers, current smokers was fifty-two point four percent. That prevalence in the U.S. white male population used as comparison was fifty-one point five. The prevalence of past smokers and nonsmokers was again pretty much identical.

So smoking by itself can't account for the observed lung cancer mortality. That's not to say that there is no, that in this population the interactive effect which has been demonstrated again and again is not in operation. I think that's likely very true.

There ought to be a lot of discussion of comparisons between other studies, so I think I'll reserve that for questioning and just respond to them.

MR. LASKIN: Thanks very much, Dr. Dement.

Can we turn to some specific questions on the study? One of the matters that you mentioned just in your introductory remarks was the shipyard exposure, I take it, in Charleston, where this plant was.

THE WITNESS: A. Mmm-hmm.

Q. I'm wondering whether, in looking at the occupational histories of the employees who worked in this factory,

Q. (cont'd.) whether there was any consideration given to any prior occupational history which, for example, may have been in shipyards?

A. Okay. Obviously we don't have information on everybody in this group. Many of them were employed before the U.S. Public Health Service questionnaires of 1964 and 1971. But we do have information on a number of the lung cancer cases.

Of the twenty-six lung cancers which we have in this group, we had past exposure, past employment information on ten. These have been reviewed and none of these had dockyard experience.

I might add another point. If the dockyard experience were to have a significant effect on this group, you would expect to have a force of in fact reversing the dose-response relationships that we saw.

For example, those who were short-term employees, had low cumulative dose, have much greater probability of being exposed in shipyards so would have a reversing trend on the dose response.

A second important point is, we only observe one mesothelioma in this group. Allison McDonald, in her Cardiff paper on this subject, her study of mesothelioma in North America, found one mesothelioma in the South Carolina shipyard workers. This shipyard used, I believe, chrysotile and crocidolite. If there were a high proportion of textile workers who were employed in shipyards, it would be, I think, pretty surprising to see only one mesothelioma in this population.

Q. The mesothelioma that you did find was a peritoneal mesothelioma?

A. That's correct.

Q. Was there any investigation of that particular case, because I have understood from at least some of the testimony

Q. (cont'd.) that we have had this past summer that there seems to be a greater preponderance of pleural mesotheliomas from exposure to chrysotile.

A. I think the literature does indicate that the pleural is more prevalent than the peritoneal, but with one case I don't think you could draw much of a conclusion.

This was a confirmed case by autopsy, so it's a fairly firm diagnosis. It was also in a long-term employee. I've forgotten the exact number of years employed, but I think it was over twenty years.

Q. Just one followup question on mesotheliomas. I note in your paper you say there were some other deaths noted as cancer of the abdomen, which you said may have been suspect. I take it in terms of their classification?

A. Yes. All the death certificates were reviewed. The problem with this disease is the way it is coded in the ICDA rubrics. All the death certificates were reviewed for any suggested evidence of misdiagnosed mesothelioma, and there were a number of cases...and I believe two at least...which mentioned cancer of the abdomen, but no supporting pathology was evident, so we really can't draw any firm conclusions there.

That's also a fairly common statement of death certificates from lung cancer as well, so you really can't say much about them.

There is perhaps some under-diagnosis as in most cohort studies which rely on death certificates.

Q. Can we turn to some of your exposure data, and can we look at table one in your paper? I guess it's at page two of the paper.

Because what struck me in looking at table number one is that in total numbers you had a large amount of environmental data. In fact, it would appear that virtually all of it is in the

Q. (cont'd.) period from 1960 onwards.

A. As you might expect in a study of this kind.

Q. Yes. Can you give us any assessment of the reliability or the extent of the data...I see the numbers here, but in more detail pre-1960, as it were?

A. Yes. I think the most important data...in fact the company began a fairly comprehensive industrial hygiene program using...taking samples on a very frequent and periodic basis in about 1956. Before that, other sources of data were available.

The company's insurance carrier conducted a number of detailed industrial hygiene studies in the plant prior to 1940, and these were well described in reports, including the methods that were used to collect the samples.

Also, the controls in effect at the time.

Attempts were made in many cases to collect breathing zone samples. A most important piece of information that gives some, I think, reasonably reliable exposure estimates in the 1930 era, is the U.S. Public Health Service study.

This was, again, a detailed study of engineering controls and exposures in the plant, and then most samples were breathing zone samples, which is a pretty unique situation in that time period.

Also, some attempts were made to actually shut off ventilation in the plant...ventilation was in fact in many operations...and evaluate exposures prior to engineering controls. These data fairly closely replicated levels measured by the insurance carrier in prior industrial hygiene studies.

So I think the two important points about the previous data, admittedly we would like to have more data, but it does represent, compared to other studies, an appreciable quantity of data.

Second of all, the exposure estimates were

A. (cont'd.) attempted, in many cases, to be breathing zone samples, and thirdly, at the time the exposures were measured, some attempts were made to describe the controls in effect during that time.

So I think you really have to look at the whole picture and how consistent are the data, and they are very consistent.

Another important point is, this is a textile operation and U.S. textile operations really have not changed a lot over the study period. In 1975, if you walk into the plant you will see the same carding machines, the same controls, the same looms that were in place in the forties.

So the same control systems were in effect during the period of time 1956, when the detailed company sampling program started, and there again remarkably consistent with the prior U.S. Public Health Service data.

Q. Were you privy to the actual measurements that were taken? I mean, did they give you the actual documentation or was it simply a report of what the exposure data had been?

A. No. The company was very co-operative in opening up their files. Most of the files were microfilmed during plant visits, and they supplied really all the data that they had in terms of the raw data reports. So it's not a summary piece of information that we've gotten. It's an attempt to actually collect every industrial hygiene sample that had been taken at the plant.

Q. Is what you are telling us that there was a fair bit of internal consistency between the insurance company data, the government data and the company's own data?

A. Very good consistency, I would say.

In fact, we have reviewed the sampling methods to make sure that the sampling methods were comparable, and samples were collected by then-recommended impinger sampling methods. I think the Asbestos Textile Institute had a recommended method that

A. (cont'd.) was a spinoff of the U.S. Public Health Service method, really was generated as an outgrowth of the 1938 study. This was a method that was used to collect all the industrial hygiene impinger samples. So there is consistency of methods of collection, and also a good consistency of levels that were measured.

Q. I take it there were not measurements made in every year between 1930 and 1960?

A. That's correct.

Q. How many years...can you give us any ballpark figure as to how many years we would be missing data for?

A. I don't have a good number on that just right offhand.

The method I used to estimate exposures, one of trying to model, use a linear statistical model to actually model exposure levels in the plant.

The reason for doing that was precisely that. Where there were holes in the data, to try to use all the available industrial hygiene data to fill in those gaps.

I think that's probably a reasonable approach rather than simply averaging, for example, rather than simply averaging industrial hygiene data for each year, to actually use all of the available industrial hygiene data, the changes in engineering controls, the changes in processes and the knowledge of jobs in a modelling process to try to predict exposures between unsampled periods.

Q. Without becoming too complex about it, as I no doubt won't comprehend it, but can you just give us an example? Suppose you had some data, say in 1938, and you didn't have any data again until 1942 or 1943, what kinds of assumptions would you make or what would you do to construct exposure estimates?

A. Maybe I could best...

Q. Sure.

A. Is there something to draw with here?

Q. There used to be.

A. If you look, first of all let me describe what was done with the data. Each...the plant was first divided into areas or zones. Each area or zone corresponded really to a physical area in the plant and in almost every case a given plant operation. So these were what I called exposure zones.

Within the zones, jobs were assigned to job categories. So for each of those zones, a separate linear model was developed. So if, for example, if I looked roughly between say 1930 and 1975, take a job, take a job category, I might have... my model might predict something that happens like this: What this would represent, this drawing, in my exposure models, I tried to document when engineering controls were put into effect so in the models the change in exposures actually...the models account for this change and the fact of engineering controls put in. It takes into account job. I have, for example, say this is zone one, which is fiber preparation, this might represent one job category. I might have four different job categories that are being measured in this way.

So I'm taking into account within my linear models job, what area...well, preparation area...job, changes in controls. So it's a linear model and the parameters of that linear model are actually estimated from the industrial hygiene data.

DR. UFFEN: What would those units be like, particles or fibers?

THE WITNESS: This will be fibers per c.c.

DR. UFFEN: When it was a long time back, would you have estimated the particles and then converted?

THE WITNESS: Well, the first thing I did was try to estimate this conversion between the two methods, and I applied that conversion to all the earlier data. So it's now in one, you know, at least one unit of measure.

THE WITNESS: (cont'd.) So what I'm doing is, I'm trying to model the plant from what I know physically happened in jobs and processes.

Your question about missing data, this is what happens. If I have...the parameter that estimates this line, it's estimated in the model, my industrial hygiene data was really used to try to come up with what that parameter was.

For example, I might have a bunch of different exposure measurements here, some here. So what the model does, it says I know there is no change in the process or control during that period of time. What is my best estimator of this gap period?

The best estimator, if you don't know anything happens, is probably data before and after. So that's exactly what happened.

MR. LASKIN: Q. If there was some engineering control change, you would make some assumption?

THE WITNESS: A. For some engineering control change, that was plugged in to the linear model and it was tested, in the models we actually tested, whether or not this was a significant change in exposure. If it was, that parameter was added to the model which would in fact lower the exposure.

But the key to it all in terms of these unknown periods is trying to make sure you have them in the plant. This was a pretty painstaking process of going through engineering records, insurance company records, U.S. Public Health Service records, state employee health records and also discussions with long-time employees in this plant - engineering employees, specifically. The dissertation itself, the first chapters of that, are in fact an attempt to document those operations and changes.

I might add also that the process that we've gone through in these exposure estimates is one of sort of a double-blind process. The exposure estimates and the descriptions of

A. (cont'd.) process actually took place about a year before any of the mortality data were collected.

I developed the descriptions of the operations and controls and sent it to the company for their review, modifications, suggestions, to make it as correct as we could. After I received that back, the statistical models of the actual environmental data were analyzed, the exposure models generated. That again was sent back to the company for their review...again, prior to any industrial hygiene data.

So we were both blind in terms of having any knowledge of the mortality outcome. I think it's pretty important.

Q. Can we turn to your conversion estimates, and as I take it from your paper and what you said to us earlier, you basically had two sets of data - U.S. Public Health data for 1965, and company side-by-side sampling for a three-year period, 1968 to 1971?

A. Yes, that's correct. That's correct.

Q. Did you then...whatever conversions those data produced did you then just simply plug them back in to your exposure estimates in particles over time?

A. That's right. What happened was, once this conversion was in fact estimated, a file was generated...a computer file was generated that contained each of the individual measurements, and the conversion simply...if it had one million particles per cubic foot exposure by impinger, and was in one of the operations other than preparation, was multiplied by three to come out with a level in fibers per c.c.

That was the file that was used to estimate the parameters of the linear model.

Q. Were there side-by-side data for every job that...every job description that there was in the plant, or every exposure area that there was in the plant?

A. There were, in the textile operation...you must remember that this plant in addition to textiles also has or has had a number of different process through the years, but textiles has been its mainstay...some rubber-coated materials in another building.

Fortunately...now, let me digress a bit...fortunately there was seniority system in place that was fairly separate between the two operations. I mean seniority in terms of bidding for jobs. So that there was very little overlap between textile workers and workers in the other plant operations, and in fact reviewing the work histories of the twenty-six lung cancers, I find in fact only one of these ever had any periods of employment at all in anything besides...any of the other plant operations.

So within textiles, yes. There was a reasonable amount of data for each one of the plant operations.

Q. Entry to your cohort, I take it, was a minimum six months employment in textiles, and one month between...one month employment between 19...

A. You had to have been employed in textiles six months, and at least one month between January 1 of 1940, and December 31st of 1965.

Q. Then you did your followup period of 1975?

A. Yes. The reason for allowing, stopping entry into the cohort in 1965, was to...for everybody in the cohort have a minimum latency or period from first employment of ten years. So if a person entered into the cohort in 1965, he would, by 1975, have accomplished at least ten years of latency.

Q. What kind of employment duration distribution did you get using a minimum six months employment in textiles? Is this...is it a fairly transient worker population or is it a long-term population?

A. Its, I think, characteristic would be both ways. It's pretty difficult in most industrial operations where

A. (cont'd.) you do have quite a bit of turnover initially among new employees, but those who stay past the period of four or five years become fairly stable employees.

In those dose-response analysis, the attempt in the dose-response analysis was really the way the dose categories were chosen. This was again done blind. Dose categories were chosen without knowledge of where cases might fall in there. It was done in an attempt to try to come up with at least three dose categories in which person-years of observation would be reasonably consistent. So ...

Q. I take it we can get that from looking at table seven in the paper?

A. You can look at the observed deaths for lung cancer, which shows that these were fairly consistent.

Q. At your dose categories?

A. Right.

This is how dose was calculated in my studies, in fibers per c.c. times days, because the work histories were in fact recorded down to the day that a person changed jobs.

Q. I was going to ask you why they were expressed in terms of days.

A. I might add also that the conversion of fiber c.c. days to fiber c.c. years in figure two, it, in my estimation of cumulative exposures, the work week...no account was taken, for example, for weekends.

So what I've done, if a person works in a textile operation for so many days, that number of days is multiplied, including weekends. So probably we are overestimating his actual cumulative exposure by a bit. Without knowing his work schedule, this is really an upper limit.

If he never worked in textiles, he may work forty hours a week. He may not work...he doesn't work seven days in most cases.

Q. You are saying that when you made the conversion from days to years, you assumed a seven-day work week?

A. That's right.

Q. Whereas in fact if it had been, if one assumed a normal five-day work week, or even a six-day work week, you've got a slight overestimate?

A. Mmm-hmm.

DR. UFFEN: More than a slight overestimate.

THE WITNESS: Well, it's two-sevenths. That chart, that line, as you say, is actually based on that.

MR. LASKIN: Q. We are now looking at the end of the paper, at figure two...the last, the very last diagram in the paper?

THE WITNESS: A. Yes, figure two.

DR. UFFEN: Do I understand you used three hundred and sixty-five days?

THE WITNESS: To come up with fiber c.c. years.

DR. UFFEN: All right. And if anybody didn't like that, they could use...

THE WITNESS: They could use whatever they liked.

DR. UFFEN: I beg your pardon?

THE WITNESS: They could use whatever they liked.

DR. UFFEN: Two hundred and seventy, three hundred, whatever you wanted to. But you used three sixty-five. Okay. I understand.

MR. LASKIN: Q. In fact, I take it you get a steeper line?

THE WITNESS: A. Well, what it would do, it would shift...it might shift some of the observed deaths to lower dose categories.

DR. UFFEN: Lower?

THE WITNESS: Lower.

MR. LASKIN: Q. Was there any particular thinking behind using three hundred and sixty-five days as opposed to Dr. Uffen's two-seventy or three hundred?

THE WITNESS: A. Whatever you use is going to be pretty much, you know, a judgemental thing, because workers do have overtime hours, they work on weekends, so using the highest number represents a conservative estimate.

DR. MUSTARD: Counsel...

MR. LASKIN: Yes, Dr. Mustard.

DR. MUSTARD: Could I ask a question about table seven?

MR. LASKIN: Yes.

DR. MUSTARD: Can you tell me the size of the cohort for each of your exposures among your original seven hundred and eighty workers, or thereabouts?

THE WITNESS: Well, I have to remember that the way person-years...the way that expected deaths and dose response is analyzed in this cohort.....a person worked his way from the lowest dose category to his highest dose category during the followup period. So his person-years, for example a person who died in the highest-dose group, will in fact have worked his way through the lowest-dose group and added person-years in that group.

DR. MUSTARD: So then he's part of the whole business?

THE WITNESS: That's right.

DR. MUSTARD: Is that what you are saying?

THE WITNESS: Yes.

DR. MUSTARD: I would appreciate it if you could explain it, because I want to make sure that I'm counting the same person twice or just once.

MR. LASKIN: I wanted to ask you that, too.

THE WITNESS: Person-years are determined in...

MR. LASKIN: Q. In all exposure categories?

THE WITNESS: A. Well, to the one he gets to, in the highest group.

(REPORTER'S NOTE: At this point Dr. Dement draws an illustration on the board.)

THE WITNESS: Let this be A here, and just arbitrarily say this is a five year age group from twenty through twenty-five, going down to seventy through seventy-five. This will be calendar time... 1930, or 1940 in that case, 1975.

What was used in this thing as a concept of a person-year, a person who lives over one year is a person-year.

So a person who enters my cohort in 1940, say he's twenty years old in 1940, he works his way down the diagonal in this group, so if he was twenty in 1940, he would put five person-years in this cell, he would put five in here, he would... whatever faction, if he died, this represents the fact that he has died.

So person-years were in each of these cells.

The way that the cumulative dose thing was done, I had a number of these different tables similar to this, so this might represent dose group one.

I have another table that would have the same stuff in it...I have it here...that would still be calendar time in age.

Okay, a worker enters this cohort. During this whole time he has contributed person-years, his cumulative exposure has also been increasing. So this is less...this group is less than ten thousand fiber/c.c. times days, and this is the next dose group.

What happens, during the time this worker is moving through this diagonal of this matrix, when his exposure, his cumulative exposure, becomes more than this dose category, he jumps

THE WITNESS: (cont'd.) over to another table. He jumps over to this table.

He would enter this table at the age that he was here and at the calendar time, so he would actually enter someplace down here and start to contribute person-years in this dose group.

So during his lifetime he actually moves from one dose to another, contributing person-years to each of these dose categories.

Okay, he dies. Say he dies in this group. In order to come up with the observed and expected deaths for each of the dose categories, death rates are applied and in this case, U.S. white male death rates, specific for calendar time and age, are applied to the person-years here. So we come up, if you add them over all these cells, you have some sum of deaths.

You also have some observed deaths that happen over here, so it's observed and expected. You come out with your SMR for this group.

DR. MUSTARD: And when he jumps to the next cell, you just keep on accumulating...

THE WITNESS: He keeps moving his...

DR. MUSTARD: ...sectors.

THE WITNESS: Right.

These person-years were just stopped when he moved over here. He no longer accumulated person-years in this group. He only accumulates in this group.

When he comes to the highest increment here, he'll move on to the next table. But there were four dose groups in that paper.

DR. MUSTARD: But your expected mortality calculations that you had based on being in that cell has to be flipped over with him into the next cell.

THE WITNESS: Now, this is...he has an overlapping

THE WITNESS: (cont'd.) followup period, an exposure period. This is a fairly common method. It's been used to look at dose response.

I guess the question is, what best models what actually happens in life.

DR. MUSTARD: Another question about this thing, I can assume looking at this table that I could have entered the plant in 1940, and operated in a sector which had low fiber exposure, where a colleague could have entered the plant into a sector with a high fiber exposure.

THE WITNESS: Right.

DR. MUSTARD: We both could have gone through the plant together. The high fiber exposure could have kept dropping as I aged in the plant and controls came in. I would come out with a different cumulative dose than the person that came in at lower fiber exposure.

THE WITNESS: That's right.

DR. MUSTARD: I guess my problem is then, if I was concerned about initial dose effect and latency, and whereas I want to get a question about the difference in outcome for people might really be dependent upon a twenty year latency period and I really want to know what the exposure is during that critical initial period, I can't really get that evidence out of this because of the way it has been handled. I'm only looking at averaging phenomenon and I really can't ask the question of what about variation in exposure at the point at which you start, rather than cumulative exposure.

Am I making myself clear?

THE WITNESS: I think that is something that has been the subject of a lot of discussion in the epidemiology literature, but your question is, I guess, two points: How do you account for exposure level - somebody who works for twenty years

THE WITNESS: (cont'd.) at one given level - and your second question was latency, or a point at which the cancer is an issue.

DR. MUSTARD: Suppose I want to test this hypothesis that we have had, if I understand the comments of gentlemen correctly, presentations that said it isn't so much your age at which you are exposed in terms of susceptibility, it's the time you live after exposure which is the determination of the outcome.

THE WITNESS: Okay.

DR. MUSTARD: And therefore, if I want to test my hypothesis, if you get a dose of fiber exposure at age twenty over a period of six months, and you are going to live twenty years, you are going to have an outcome that could be to a large extent related to that initial exposure.

THE WITNESS: Yes.

DR. MUSTARD: So I would be anxious to know what those exposure levels are to try to get some estimate about them.

THE WITNESS: Okay. What I've done...if you look on table seven, I think I can answer your question. It says, summary of dose-response relationships for selected causes among white male workers after fifteen or more years latency.

So what I've done, each of these groups has to have at least fifteen years latency, so I've tried to take into account your, I think, your first question, that as you go through this table you don't have to acquire latency very early, you achieve it after some point down the line.

So in fact what happens, this is going to sort of complicate it, this is actually a three-dimensional matrix. You don't know the dimension back here as latency. Computers don't mind. It becomes sort of messy on boards.

What it is, in addition to a person moving across this diagnonal, if you took latency here on this diagonal - zero

THE WITNESS: (cont'd.) years to twenty years, say, he is also moving along that diagnonal in the back, okay? But he jumps over here, he starts at the same point that he was. If he was at fifteen years latency when he moved over to this next cell, he begins picking up latency here.

In order to account for his lack of risk in the first fifteen years, in each dose group person-years were actually started when they achieved a fifteen-year latency.

So each one of the exposure groups has a minimum of fifteen years latency.

DR. UFFEN: Is the definition of latency just time since first exposure?

THE WITNESS: Well, in the epidemiologic literature it is, because we don't necessarily have a point in time when the process was initiated. So we have to back up and use that as a surrogate relative.

DR. UFFEN: That's what you are using here?

THE WITNESS: That's right - time from first employment in the industry.

DR. UFFEN: First employment?

THE WITNESS: First employment in this...

DR. UFFEN: ...in this place?

THE WITNESS: Yes.

DR. MUSTARD: But the cumulative dose still is truly cumulative dose, taking all these other things into account. It can't answer the question of peak...well, I'm even going to be more specific. Initial exposure during the first year, you can't really...that we can't really pick out of this particular data?

I guess nobody has done it.

THE WITNESS: It becomes a nightmare to try to handle it with this type of cohort.

I guess the question of...it really gets down to

THE WITNESS: (cont'd.) the question of what I call wasted dose. For example, in this highest-dose category we have accumulated a person's dose throughout his working lifetime. Those last ten years may have been meaningless as far as his eventual outcome of lung cancer. He may have already had the process in this year.

So there is potentially, at least in the high-dose categories, some wasted dose, which...the effect would be to make the dose-response even more steep, surprisingly enough.

But the other thing is, what are you going to use as your measure of lifetime exposures, and I've chosen cumulative exposure concept.

I think for materials like asbestos where it does have a reasonably long residency time in the lung, probably cumulative dose is a reasonable index of exposure.

I don't have any real way of taking into account peaks in this study. I know that there are some peaks, and there are also some valleys. I have chosen to use an average exposure level.

MR. LASKIN: Q. Is the effect of moving people through your categories and accumulating person-years, is the effect to tend to underestimate the risk at the lower levels and overestimate it slightly at the higher level?

THE WITNESS: Well, potentially, because what you have...a person who achieved a high-dose category, the process that was eventually going to achieve his cancer, was going to develop or cause him to develop his cancer, in fact may have been initiated back in two dose categories before. So he had accumulated exposure and you have attributed that death to the high-dose category when in fact you may have, maybe should have attributed it to a lower-dose category.

So the overall effect is probably one to underestimate

A. (cont'd.) the risk.

Q. Also, if he had died immediately upon entering a particular exposure category, he wouldn't have accumulated any person-years for the calculation in that exposure category?

A. Yes.

Q. What...can you explain what the thinking was behind that kind of methodology?

A. Well, it's really to try to model events as they actually happened, recognizing the problem of...you have an overlapping followup period and dose period. Other investigators, and I think you had Dr. Enterline here and I think also Dr. McDonald, in his 1965, or in his study of workers...I think it began about 1965...

Q. Who are we talking about, Enterline?

A. Enterline's mortality study.

Q. The retirees?

A. Retirees.

He...first of all he chose to study retirees. Second of all, the method that he used to assign a person to a dose category was a bit easier than what I have here, because it did not have a period of overlapping exposure and followup. His people had already ceased their exposure, so he simply assigned it to the category, the cumulative dose category, at the time they retired.

I think one problem with the retiree's study is one of selection bias into the group. You have, at least potentially have, a survivor population. In fact, if you look at this cohort, textile workers, most of the lung cancer deaths occurred before age sixty-five.

Q. From some of the studies I have looked at, another approach appears to be to have a person accumulate exposure up to a fixed point - say twenty years - then cut off your exposure

Q. (cont'd.) and then have a followup period. Do you see any problems with that approach? Do you see any advantages, disadvantages, as to that kind of methodology as opposed to what you have done?

A. There are advantages and disadvantages to both methods, I suppose.

I think one of the potential problems with that method is that in the low-dose categories you aren't contributing a person's person-years as he moves through the followup period.

For example, in a low-dose category, you would not attribute a person's person-years after...who achieved a high-dose group.

I don't know. In terms of weighing and outweighing one method versus the other, I think in fact...and I've been toying with this in this group...when you restrict it to beginning person-years at fifteen or more years latency, as table seven does in this group, you have in fact sort of done that in some ways. You've taken out a lot of those initial person-years that were attributed by the high-dose group to the low-dose group, so in essence it is practically the same method.

By fifteen years there is relatively little change of the exposure category. So I'm not saying one method is better or worse than the other. They both have their potential problems.

In fact, I think they are comparable. I don't think that methodology can really account for the differences between, for example McDonald's chrysotile group and this.

MR. LASKIN: Dr. Uffen?

DR. UFFEN: I wonder if I could ask a question about that number of days in the year, just before we get off onto something else, to make sure I understand it.

If there were to have been a consistent change

DR. UFFEN: (cont'd.) in the number of days worked per year from the early days, twenty years or thirty years ago, to now, in the direction that the men worked less in recent years, does that suggest to me correctly that the contribution of the most recent years to the cumulative exposure is overestimated?

THE WITNESS: If you say relative to...

DR. UFFEN: Relative, yes.

THE WITNESS: ...relative to the other dose groups, that's perhaps true. But in fact they are both overestimated for actual exposures.

DR. UFFEN: I beg your pardon?

THE WITNESS: They are both overestimates of actual exposures.

DR. UFFEN: But the amount of exposure in the early days may have been more intense than since improvements were introduced?

THE WITNESS: Mmm-hmm.

DR. UFFEN: So that if you overestimate the more recent years, it's not a linear relationship - it's not the same as overestimating the early years by the same amount, because of the higher exposures.

THE WITNESS: But on the same token, I've used the worst-case case, and I..in total, it's overestimated.

DR. UFFEN: But what I'm getting at is, does this affect the linearity? You plot three points - admittedly you haven't got much data, I'm not criticizing that - and it looks like a straight line is about the best, and then if you find that there's reasons for wondering whether the more recent data has been overestimated, that would change it from a linear relation to something else.

THE WITNESS: I don't think so. I think it will have the effect of shifting the relationship, not affecting the fact

THE WITNESS: (cont'd.) whether it's linear or not. I think it may affect the slope, but not the shape.

DR. UFFEN: It would stiffen up the slope?

THE WITNESS: It would make it more steep.

DR. UFFEN: Than you've got in figure two.

Okay, thanks.

MR. LASKIN: Q. Can I just ask you one further question about your conversion, which I haven't asked before?

I had understood from some other testimony we heard this summer that two people called Eyre and Lynch in the United Stated had also attempted to do some side-by-side sampling in a textile factory, and I take it it wasn't the Charleston factory, but another textile factory, and had come up with some conversion ratios which seem to be higher than three-to-one.

THE WITNESS: A. Well, in fact the Charleston plant was one of the plants that was studied.

Q. It was?

A. Eyre and Lynch have...they studied a number of different plants. If you look at the data from the Eyre and Lynch study, and it does at least identify plants in some of their papers, there is more...there is as much variability between plants as there is between operations within a plant. In fact, sometimes more.

So although this data may be on the low side of their range of conversions, it certainly is within the range of their conversions and it is specific of this plant.

Q. Did you look at their conversions for Charleston, along with...

A. It's in their paper. I tried to identify the Charleston piece in their paper.

The problem with...it's discussed in their larger text, trying to compare those. In fact, there is a table of

A. (cont'd.) comparison.

The Eyre and Lynch actually, there was a lot of discussion of conversions between counts of fibers longer than ten microns versus impinger, and that was published...in fact, a published data.

I have tried to, in page 102, table three dash two, I have tried to compare what they found.

Q. You are talking now about your thesis?

A. Right.

Going back through their publications and trying to compare, and this is sort of a range of conversions that I came up with, using actually data from two of their papers.

It's pretty much within the range of mine. In preparation, I'm on the high side. In the others I'm, I would say except for carding, I'm well within the mid-range of most of those.

In fact if you look at one other piece of information on conversion, was the Lyon paper that McDonald presented on his preliminary analysis of data for Quebec, and he, I think, found an average conversion of roughly three for the chrysotile miners and millers.

Q. Did you put confidence intervals on your conversion ratios for each point of operation?

A. I put the confidence intervals on the mean conversion ratio. That's given in the paper.

Q. At figure one?

A. Yes. There's considerable overlap between the two methods for everything besides preparation, and for that I chose to go with the higher conversion.

Both intuitively, because one of the functions of preparation is to remove short fibers in textile operations.

Q. I suppose one of the...I appreciate your point

Q. (cont'd.) that your ratios are certainly within the Eyre and Lynch estimates, but I'm just looking at the estimates. Their range for fiber preparation is one point eight to nine point one. That's where you used eight.

A. Right.

Q. But their range for carding is three point eight to eleven point three, and for weaving, two point one to eleven point six?

A. Mmm-hmm.

Q. And you used three for both of those?

A. Mmm-hmm.

Q. It seems to me, without doing all the mathematics, that the midpoint of both carding and weaving for both Eyre and Lynch is higher than their midpoint for preparation?

A. One comment is, I think the comment about variations by plant being more than the variations by operations. I think you have to look at specific plants.

Second of all, the distribution of ratios, these are average ratios. These are average ratios for the four plants.

That distribution of ratios is not normal at all. It's very much skewed with a long tail to the right, so to use an average of an average is probably not the way to do it. You want some better measure of central tendency in that distribution. That's probably closer to a log normal than a normal distribution.

So you are probably best using a median ratio rather than an average, and that median is going to be very much pushed down to the lower side.

I don't think you want to use averages.

Q. In coming to your three for all operations other than preparation, did you lump all the other operations in together? How did you arrive at the three? Was it done by a separate breakdown of each of the other operations?

A. It was done by, the analysis of both sets of data was done first trying to take into account each textile operation, trying to look for differences of conversion factors between textile operations. So independently, looking at differences of conversions, with the paired-sample data the data did not indicate any statistical differences between the plant operations. For the concurrent sample data, 1968 to 1971 data, where both measures were used, it was significant, the preparation operation.

So yes, there was some attempt to look at differences of plant operation before in fact coming up with one or two conversion estimates.

I guess what I'm saying is, the textile operations were all analyzed separately. Separately in the sense it was done with linear models which took into account operation.

Q. Can you give us a general assessment, in your own point of view, as to the significance of your findings in relation to other cohort studies?

A. This is one...epidemiology is one of replication. I would like to see the study replicated. I think you have to take it and look at the overall data and assess in terms of a lot of different parameters. First of all, is it consistent with the body of other epidemiologic data, and it is in a sense that here is a chrysotile operation and we have seen increases in respiratory cancers and asbestosis. The steepness of dose-response data is different, very much different from the McDonald studies, not as different...not that much difference between us and Enterline as the McDonald.

I think a couple of points..first of all textile operations are not the same as brake lining operations, they aren't the same as mining and milling operations.

We, in some other work, have attempted to look

A. (cont'd.) at the airborne fiber size, dimensional characteristics of textile versus other operations using chrysotile.

I think two things - first we find that in textiles and the nature of the operation, because you need them, you find a prevalence of longer, thinner fibers. From the animal bioassay data, these appear to be the most important in terms, at least, of cancer outcome, from the models that have been used, implantation models.

So I think it's quite likely that we will see differences by industrial sectors. I think that's almost given that, knowing the characteristics of exposure.

Q. You are saying that what are thought to be the more dangerous fiber dimensions are more prevalent, percentage-wise, in the textile operations...

A. That's correct.

Q. ...than we might find in other kinds of industrial or mining operations?

A. It certainly is, if you compare friction products for example, with textile.

What I'm saying, I guess, there is a fraction, there is a portion of the airborne dust, given that the level is X, there is some portion of that in every operation that is biologically more active. From the data that is available, at least comparing the size characteristics, textiles appear to have that greater proportion of active material.

So, you know, what I'm saying is that, don't throw away the rest of the epidemiology in comparing this with other plants. I think it's more appropriate to look at reasons why. Is there differences in methodology that account for it, and from my assessments between us and McDonald, I really don't see that that's a big factor.

A. (cont'd.) The Enterline study, I feel there is probably underestimated risk because of the retiree population.

The next question is, what are the differences in actual exposure characteristics and levels.

Q. I take it in addition to saying don't throw away the other epidemiology, I take it you are also saying be careful about extrapolating from the results of your own study into other asbestos operations?

A. I think you have to look at...you should do it very carefully, because I think there are a lot of unknown parameters in that extrapolation, beyond just what we measure by this crude...and it really is a crude method of measuring exposure, the impinger was crude, the membrane filter method is still very crude and we are actually only looking at a very small portion of the airborne dust.

So I think before you use that measure as a common measure you've got to look very closely at the characteristics of the fiber.

MR. LASKIN: I'm sorry, Dr. Uffen.

DR. UFFEN: This raises the question in the paper that we have been discussing now, there is very little mention of the fiber length question.

THE WITNESS: Yes.

DR. UFFEN: Yet in some of your other excellent papers...I was just trying to figure which one it was here, I haven't got the number...where almost the whole paper was devoted to the analysis of the dimensions. Was it the wool one?

THE WITNESS: Yes, there were a couple.

I might add that the plans for this study, we looked at the zero/six month group and now...we looked at the greater-than-six-month in this publication...now we're completing an analysis of the one-to-six month exposure group. In addition to

THE WITNESS: (cont'd.) putting together that paper on mortality, for publication, there will also be a paper that describes the method of estimating the exposures, and the characteristics of the dust.

This was covered...the work for the dissertation in fact did look at that in quite a lot of detail.

DR. UFFEN: Your thesis?

THE WITNESS: Yes.

DR. UFFEN: But not in the paper?

THE WITNESS: It's just a matter of just not enough room in one paper to put all that data, so it will likely be in two separate, but companion, papers...to summarize those data in the dissertation.

MR. LASKIN: Q. Is the present paper that we've got in draft? Do we now have a draft..

THE WITNESS: A. The present paper is stamped draft simply because it's still in press, from the Inhaled Particles Conference, and that's the only reason. It's in press now. I don't know the exact date that it's going to come out.

Q. It will get published as part of the papers that were delivered at that conference?

A. Yes, it's part of the copy of the paper that you have.

DR. DUPRE: I was wondering, Dr. Dement, if I could just ask a question so that I could perhaps better understand your cohort in relation to the plant.

Now, your seven hundred and sixty-eight white males were all employed prior to 1965, for six months or more?

THE WITNESS: Yes.

DR. DUPRE: And going back to 1940. That's a twenty-five year period. Now...and of course they were in the textile operations of the plant?

THE WITNESS: Yes.

DR. DUPRE: Now, on page nine of your paper, you pointed out that a rough guesstimate of the total number of employees in that plant prior to 1965 would have been ten thousand.

THE WITNESS: That's right.

DR. DUPRE: Now, do I take it that of course a substantial number of employees in that plant would be involved in nontextile operations?

THE WITNESS: That varied. The plant has had a history of change in products as the need arose, but there were in some cases a substantial number of nontextile.

The other point, I suppose, this is the more than six month.

DR. DUPRE: That would be your black textile employees as well?

THE WITNESS: Yes, everybody.

DR. DUPRE: Who would be in the ten thousand...

THE WITNESS: That's right. Blacks, males and females.

The female population of this group is nearly as large as the male, and I am currently working on this group, as is Allison McDonald. I think Allison reported some of the data recently.

The problem with the females is that the followup is just very difficult, with changes of names. You just can't locate them. So we haven't published that data as yet.

Preliminary analyses of the data, making some very conservative assumptions as to those who we don't know about, assuming that they are alive, still indicates the excess of respiratory cancer in the females as well.

But the ten thousand includes everybody - textile, nontextile, black, white, male and female.

DR. DUPRE: So at this point...

THE WITNESS: I might add also, it includes those who might have been employed before 19...

DR. DUPRE: Yes, before 19...

THE WITNESS: Which is, again, the plant began in the late 1800's, they also get a substantial number.

DR. DUPRE: Of course another point of reference to your cohort is that the ten thousand include all employees who were employed for six months or less, and your high turnover among new employees might account for all of that...for a lot of it?

THE WITNESS: There's a substantial turnover and if you look at the size, for example in the white male group, those who were less than six months and more than six months, they are about equal.

DR. DUPRE: Now I take it, just having asked these questions as background, that you have reasonable confidence that the seven hundred and sixty-eight white males employed for six months or longer between 1940 and 1965 are pretty close to the universe of white males employed in that plant for six months or longer between 1940 and 1965?

THE WITNESS: As reasonable as one can be, and that's largely based on discussions with plant personnel and the personnel office...as far as completeness of those records.

And those...when you find a large prevalence of people in the plant personnel records with less than two weeks of employment, you have a good feeling that if you moved up to six months you really do have a fairly complete group.

DR. DUPRE: Did you write down your seven hundred and sixty-eight in terms of those who had been employed six months to five years, five years to ten, ten to fifteen?

THE WITNESS: There is a table, at least in looking at outcome...

MR. LASKIN: Table five, I think.

THE WITNESS: ...by duration of...

MR. LASKIN: Tables four and five?

THE WITNESS: Table five, yes. Yes, table four. Because, you know, less years of employment, I just broke it down into four categories here, to look at...

DR. DUPRE: These, as I gather though, are all the years of employment among your employees who died?

THE WITNESS: No, this is of everybody. The expected deaths are based on person-years for everbody.

DR. DUPRE: Oh, I see. Fine.

THE WITNESS: I might add, preliminary data on... and I say it's preliminary...it's pretty well followed up in terms of completeness for this one-to-six-month group...follows the pattern that was at least set by the other dose-response data in that I have, as you might expect, in terms of cumulative dose I now have a group that has a very low lifetime cumulative dose.

As you might also expect, they have a very low lung cancer SMR. I think...don't quote me on the numbers...but I think it's four observed, five observed and roughly four expected. So it's not significantly excess, but it still does follow the same pattern that was established.

It gives me a little more confidence in the reference population, too, in terms of observed and expected.

MR. LASKIN: Q. Just to follow the chairman's questions, I take it from table four your expected calculation is nonetheless based on the same person-years methodology that you told us about before, so there isn't a breakdown of actual number of employees of the seven sixty-eight who worked six months to five years, who worked five to ten years and so on, in terms of the actual individual, and does that kind of data exist...

THE WITNESS: A. It does exist and it's just not put in here.

Q. It's just not reflected?

A. Yes, it's just not in this paper. The data does exist. I can't recall offhand what it really is, but...

Q. I'm just wondering whether you have any sense of whether there were a lot of long-term employees in this cohort of seventy sixty-eight, or not very many?

A. That's pretty much the distribution you would expect. When you look at your expected deaths by years employed, you see that most of them were in the less-than-ten-year group, and that's where most of your employees lie, less than ten years. I would say roughly three-fourths of your employees lie in that group.

Q. Can I just ask you one or two more detailed questions?

If we go to table eight, which is at page eight of the paper, and this relates to your use of a reference population, which was the U.S. population, and I followed your argument on why you chose to reject using the actual local county rates.

Can you...and I'm not sure it was in the paper...can you explain the thinking behind rejecting use of contiguous county rates?

A. Well, even with contiguous counties you still have some effect of the local shipyard. They didn't draw all just from one county.

The other problem is when you start breaking up your death rates by five-year calendar time and age groups, unless you are really going to expand that thing out and get a large number of contiguous counties, these death rates are going to be based on very few observed deaths in many cases, and the stability of death rates becomes a major issue.

There is not much advantage if you are going to expand out and use a large number of populations, not to look at the state as a whole, and in fact that death rate is equivalent to the U.S., and Allison McDonald in her analysis did use state death

A. (cont'd.) rates and her lung cancer SMR's, not surprisingly, were fairly consistent with mine.

I think there is good reason to at least expand out to a much broader base than contiguous counties, both from an influence-of-the-shipyard point of view and from a statistical point of view as well.

Q. Can you give us any general assessment of the affect of chrysotile as compared to the affect of other fibers, in your understanding of the various cohort studies? I'm thinking of your high SMR's for lung cancer on the one hand, but your finding of only one mesothelioma.

A. Well, chrysotile and mesothelioma has been a subject of...well, I won't say considerable controversy, but as I look over the bulk of the data, I could come to a conclusion that yes, chrysotile does cause this disease. Animal data where the material is actually put at the site does produce the disease. It's consistent.

When you look at the industrial population studied exposed to chrysotile versus mixtures of chrysotile and the amphiboles, you find that the percent prevalence or the percentage of all the deaths attributable to mesothelioma is markedly different in chrysotile versus mixed-fiber exposure situations.

That's consistent and my data are consistent with that.

Then you probably, as many people have talked about and I think Dr. Wagner is probably an advocate as well, chrysotile airborne characteristics and the characteristics of the fiber after it's deposited are probably fairly curvey, tensile strength is probably not quite as much as the others, and it doesn't deposit and it doesn't migrate as easily to the pleural cavity.

I don't disagree. My data has to support the

A. (cont'd.) conclusion that, at least as far as chrysotile is concerned, it's less associated with this disease.

Although I think it's probably not as low as my cohort has indicated, perhaps because of underdiagnosis.

Q. You say the reconcilation between the epidemiology and the animal studies on the other side is that in the animal studies you actually put the chrysotile at the specific spot and ...

A. That's right.

Q. ...in reality there are difficulties in getting to the spot.

A. When you put it there artificially, it produces a large tumor yield, equivalent to most of the others. But the epidemiology doesn't bear out that fact in populations.

Artificially...it has been artifically put at the site when it actually by inhalation has to make its way first to be deposited in the lungs, and second of all, after it is deposited, available data, at least, indicates it has to migrate to the pleurae.

DR. DUPRE: Dr. Dement, from your knowledge of the animal experimentation, what do experiments by inhalation as opposed to injection suggest in terms of fiber type differences?

A. Well, by inhalation all fiber types have been capable of producing lung cancer.

DR. DUPRE: I'm thinking of mesothelioma in particular.

THE WITNESS: It's very difficult, and when I look at the animal data I know there have been a few...Chris Wagner being more successful than others at producing mesothelioma by inhalation. At first, I guess, you've got to wonder by inhalation whether the rodent, his respiratory deposition and clearance, is reasonably...whether that's an adequate model for human exposure.

THE WITNESS: (cont'd.) I know chrysotile probably has, in the rodent studies, a possibility for deep lung penetration is not very good.

So first of all I wonder about the model itself. But, all types have produced it. Chrysotile, I believe, has produced fewer by inhalation, fewer mesotheliomas by inhalation, than any other type.

To me that's consistent with the human data.

MR. LASKIN: Can we take about ten minutes coffee break?

DR. DUPRE: Certainly.

Might I just perhaps ask about the geography of the day?

MR. LASKIN: Well, that's why I want to take the break.

DR. DUPRE: Among other things, do I gather that our guest must be delivered to the airport promptly at four-thirty? Or four, or three-thirty?

MR. LASKIN: I expect four.

DR. DUPRE: So we will be breaking at four.

Perhaps another thing we may want to think of is rather than break for lunch between one and two as we normally do, we may want to break at maybe twelve-thirty or one-thirty, or something like that.

M. CASGRAIN: I thought you were going to say not break at all.

DR. DUPRE: Counsel, I'm always open to wise suggestions. I'll certainly take it under advisement.

M. CASGRAIN: I'm not suggesting it!

THE INQUIRY RECESSED

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THE INQUIRY RESUMED

MR. LASKIN: Thanks, Mr. Chairman.

MR. LASKIN: Q. Dr. Dement, can I turn just briefly to one of your other papers, and I wanted to ask you a question about it in conjunction with your paper at tab nine.

I'm looking at tab eleven, which was the asbestos work group memorandum or report to Dr. Robbins and Dr. Bingham, and I take it you were part of that asbestos work group at the time?

THE WITNESS: A. Mmm-hmm.

Q. On page two of tab eleven, on page two at the bottom, in the last paragraph, there is the statement, "On the basis of available information, the committee concludes that there is no scientific basis for differentiating between asbestos fiber types for regulatory purposes."

Can I fairly ask you whether you personally subscribed to that view at the time?

A. Yes.

Q. Has your view changed in any way in light of your own paper, which came, obviously, subsequent to the report at tab number eleven?

A. Yes. The data for my paper came after this document was prepared. But the data from my paper aren't really much different from the data that was available at the time of this in terms of the mesothelioma risk by asbestos workers.

I think the group was faced with data that indicated that all types of asbestos were probably related to lung cancer and asbestosis.

Available data at the time really didn't indicate that large a difference between dose risk, dose for dose, than the other types of asbestos, amphibole types.

I think additionally the group looked at the

A. (cont'd.) contribution to overall risk to asbestos workers that mesothelioma really is, and my own opinion... I might state this is my own personal opinion...a lot of, there has been a lot of attention to that one particular element of risk when in fact it's a relatively, on a proportionate basis, a small portion of the overall risk, asbestos overall risk, when you look, in addition to mortality, you look at morbidity.

So that was, I think, some of the reasoning behind this group's rationale for not recommending differences in levels for fiber types.

DR. DUPRE: Can I just pursue one point on that, Dr. Dement? I certainly can take it that mesothelioma is indeed a small portion of overall risk in terms of mortality, but when I examine some of the studies I have been struck, perhaps wrongly, but I have been struck by the age of mesothelioma victims at time of death, which at this point, if I start to think of mesothelioma in terms of denial of life expectancy, that gives it a considerable boost on my priority structure.

Have you considered that?

THE WITNESS: Well, we were dealing in this case with a material that I think is accepted by almost everybody as being a carcinogen, and with the knowledge that...you know, at what level with a carcinogen do you have no excess risk. So I think the philosophy that was the basis for the recommendation was lowest feasible level of exposure, and we went with the philosophy of lowest feasible level, which should be protective, I suppose, to some degree for mesothelioma and for the other types of asbestos disease.

I think it was more the philosophy of the group and not so much looking...I mean, more a philosophy of looking at the overall risk than looking at one element of risk.

I wouldn't deny that mesothelioma is, for those

THE WITNESS: (cont'd.) who develop it, it's a life-shortening process. But on the other hand, asbestosis by itself, nonmalignant respiratory disease, also is a source of considerable life shortening and decrease in the quality of life.

DR. DUPRE: But I guess just one other question, if you will indulge me, counsel?

MR. LASKIN: Sure.

DR. DUPRE: If one wishes to pursue the lowest-detectable-risk philosophy, at this point if one is a member of that school and at the same time, let us say, a member of the fiber-type school, one might, of course, simply couple a lowest-detectable-risk standard with, let's say, just the outright banning of a particular type of asbestos...say crocidolite.

What is your feeling on that?

THE WITNESS: I can take that one step further. The lowest detectable risk is when you eliminate all unnecessary sources of asbestos, and that's what we recommended in this thing. Rather than look at one fiber type and say well, you get rid of crocidolite, our philosophy was - substitute where you can.

MR. LASKIN: Q. Did your group give any consideration to looking at the question of regulation industry by industry or operation by operation? Did it look at the question of distinguishing the mining situation from the textile situation?

THE WITNESS: A. This was actually a report that went to the Occupational Safety and Health Administration, which in our country is for general industry.

There is another portion of the overall assessment. NIOSH is now underway with that assessment. They are looking at the risk for mining.

The situation in mining is one where you have, in some cases, a material...there is a lot of controversy about what you call it. Do you call it asbestos? Do you call it a mineral

THE WITNESS: A. (cont'd.) fragment? What do you call it?

There is a considerable amount of controversy as to what standard you apply in that situation.

I don't have any solutions to that, but I think it's an area where a lot more epidemiology is needed, especially on mineral fragments.

Q. Are you now...what are you addressing? Are you addressing the situation of nonasbestos mines, minerals, which may throw off...

A. Well, in the United States you are going to be running...

Q. ...asbestiform fibers?

A. In the U.S. we mine relatively little asbestos, so our asbestos problems in mines is basically that. It's particularly a problem with the amphibole forms which are fairly common in some types of ore bodies...copper and the gold mine situation.

Q. I take it that issue is something you and several others addressed in the South Dakota gold mine study?

A. That was one study that was looking at the noncommercial types of fiber contamination.

Q. That study has in fact already come before this Commission, and it has come before it in conjunction with another study of that same mine done by Dr. McDonald's group, which I trust you are familiar with?

A. Yes.

Q. One of our problems is trying to reconcile what appear to be different conclusions reached by the two studies. I wonder if you can help us with that, if you could comment on the McDonald study?

A. I think one thing to recognize is that there is

A. (cont'd.) relatively little overlap between the two studies themselves...I mean in terms of people in the cohorts.

They are really studying two different cohorts. The NIOSH study cohort consisted of people who were studied in a cross-sectional medical study by the U.S. Public Health Service in about 1960, that period of time where it was studying silicosis in underground mines.

So it's defined from that group and followed from that time on.

The McDonald study was defined from a long-term employee roster. I think the number was twenty-one years or more employed.

There is relatively little overlap.

We found an excess of respiratory cancer. The numbers were small, the cohort is small.

McDonald, on the other hand, found no excess respiratory cancer, but a very large excess of silicosis mortality.

To reconcile the differences: First of all, there is at least the potential for some forms of selection bias in the McDonald study. It's essentially a survivor population.

Second of all, although not mentioned in the McDonald paper, the Homestake Mining Company operated surface facilities, and in fact operated a timbering operation in Home State and the area.

Some of these employees, I believe, were included in the McDonald study.

So if you look at his dose-response data and you look at the highest-dose category, that would be mostly those who had been underground. There is a slight excess, at least in numbers, of respiratory cancer. But not significantly excess.

So there is some differences.

NIOSH has underway a much larger study than either McDonald or our previous study of this population.

A. (cont'd.) It's a fairly...the question is very important. It has a lot of bearing on, I think, the direction that the Institute may go in a mining standard.

So this cohort is currently being studied and it's more of an attempt to go back to the overall personnel records at the plant and find a much more complete cohort over a longer period of time.

I don't have the results of that to present to you today.

Q. Is that the research project out of Stanford that we've heard about?

A. SRI was doing that under contract to NIOSH.

Q. Okay, but it's one and the same project?

A. Mmm-hmm.

There is also...that project included some more environmental sampling at Homestake Mine.

Q. Is there any idea as to when that study is going to be completed or available for public edification?

A. I'm not the project officer on it. I wouldn't want to give you a time frame on it, but I think it should be pretty quickly. That study has been going on for over two years now.

Q. One of...if we can turn, I just want to turn quickly to your own study along with Dr. Gillam et al, which is at tab four.

Another of the problems that we have been grappling with this summer, as I expect you can imagine, is whether measuring fibers greater than five microns in length with a better than three-to-one aspect ratio is an appropriate index to use, and I suppose in conjunction with that, because it's related to it, what fiber dimensions are really, quote, "the dangerous fiber dimensions", end of quote.

Q. (cont'd.) I took your evidence before the coffee break about your judgement of the textile situation, but it would appear here that there are an awful lot of short fibers.

A. That's right.

Q. Those short fibers, nonetheless, at least in the view of you and the others who did this study, are producing excess mortality.

A. Yes. I guess the question is, how short is short.

If you look at airborne dust fiber size data, you find that the portion of airborne fibers in industrial operations using asbestos can range from one percent to fifty percent, and the fifty percent is usually the amphiboles. You tend to count a greater portion of the amphibole fibers.

The Stanton data, if you look at the bioassay that Merle Stanton has published, he suggests that those fibers that are, I think less than one micron in diameter and greater than eight long, are most related to his cancer outcome.

But he also cautions that that's no magic number. That eight microns and one is no magic number to indicate that there is no risk. That's perhaps true. There is probably some gradient risk.

The problem with the phase contrast method really is it counts only a portion of airborne dust. In defence of it though, I think if we believe the bioassay data and we are in fact counting a good portion of those fibers that are longer than five microns, then perhaps it is a reasonable index of exposure.

But that's not to say that there is absolutely some cutoff of fiber size below which there may not be a risk, and I might also add that only relates to lung cancer. It doesn't really say much about the pneumoconiosis risk, although bioassay data on...by Davies and others...seem to indicate that long, thin fibers are more efficient in producing fibrosis as well.

A. (cont'd.) My own study did attempt to look at characteristics of fiber by operation, and that's presented in the dissertation.

The object of the exercise was one of...well, if I use this measure of exposure of this phase contrast method, I only account for longer than five micron fibers. Is it a valid index?

More importantly, is it a stable index within that study population?

Q. As I understand your conclusion, and correct me if I'm wrong, your conclusion was that it was a relatively stable index if you were looking at what Stanton thought were dangerous fibers...

A. That's right.

Q. ...but it was a most unstable index if you were looking at the whole spectrum of fibers?

A. That's right. That's the conclusion, because if you had to account for the differences in total fiber content by operation, that very much affects the internal stability of your estimates.

And the fact that there was at least, in terms of the dose response, some consistency, at least suggests that that measure was at least a reasonable measure of risk.

Q. Are you still a supporter of the phase contrast method, or would you like to see some other method, or one or more in conjunction?

A. Okay. I guess there is a purist side of everybody, and the person who has to be practical. For the purist side, I would like to see something more developed that would actually look at the overall exposure and be able to characterize that a bit more.

From a practical point of view, the only methods that we really have to do that are electron microscopy, and there

A. (cont'd.) are even a lot of problems in trying to generate reliable statistics by electron microscopy.

You are still confronted with the fact that you are counting fibers, and the counting variability may be as great as or greater than with phase contrast.

The other point is, I guess, from a practical point of view it's really the only thing that we have now that can be used on a routine basis for monitoring.

But I might add that when you force down levels of exposure down to point four and point five fibers per c.c, you are really...although you can measure that exposure...you must also realize that as you force that level down, the variability about that measure also increases and it increases substantially.

So at the same time that you are forcing the level down, you also have to account for that variability in your ability to enforce a standard.

You might...the other problem at that low level is mixed fibers. In an operation that happens to use glass and asbestos, and I'm at a level of point one, point five, then it becomes much more important that I be able to identify those fibers.

I think in those mixed-exposure situations, and we've touched on it in our recommendations in this, you may want to go to some backup techniques, if you will.

Q. By the electron microscope?

A. Perhaps. I'm not sure yet that, you know, to that level of sophistication all the time.

I think petrographic microscopy, polarizing light microscopy, is equally useful in some cases.

Q. In your judgement should the backup be a numbers measurement, or is there any merit to a weight or a mass measurement?

A. The problem is trying to relate the two.

A. (cont'd.) For example, if I measure X fibers per c.c. and try to convert that back to micrograms per cubic meter basis, it presents a lot of problems. So I think the backup technique really is a backup to your original analysis in terms of identification.

It should be a numbers, I think, a numbers approach.

Q. But don't you even there have the problem that the backup is going to be measuring fibers of all different sizes? You are still faced with the problem of relating, aren't you? You are relating...

A. No, no. You can measure, you know, in your backup technique look at those fibers that are longer than five micrometers and determine what proportion of those are asbestos and what portion aren't.

I think the data so far on other materials, and I say it with sort of reserved judgement, the data so far indicate that glass...at least in that size range, in the size range that we are accustomed to seeing...is not as hazardous, perhaps, as asbestos.

DR. UFFEN: Is it possible that just the latency is longer?

THE WITNESS: That has been the subject of discussion. We and NIOSH have studied glass fiber workers and another group called mineral wool workers. These were slag wool and rockwool workers.

A number of people have also studied...Phil Enterline has now underway a fairly large study and he has reported on some of it.

The glass fiber mortality study was in the oldest plant in the U.S. At the time we completed our study there were relatively few people with longer than thirty years latency.

But there was not an excess of respiratory cancer

A. (cont'd.) in the group. In fact, I think there was a small deficit of respiratory cancer.

The glass fibers...that industry is relatively young in this country. In contrast, the mineral wool industry ... and the characteristics of the fiber in terms of size are roughly the same as glass fiber, although you might produce a bit of a fine fiber in early days. But it's older.

NIOSH completed a study, and I think it's part of the package that you have, of mineral wool production workers, and again it was a mortality, and there was no...the data did not indicate any statistically significant excess overall, and it was stratified by latency and the longer latency period there was, a slight excess...at least in numbers, and again not significant... in cancer of the digestive system and respiratory disease.

But I guess that's difficult to interpret. The Enterline...the original Enterline study, and the data I think were presented in Lyon, also indicated at least some excesses in G.I. cancer with long latency in mineral wool workers.

I think that's a subject that needs considerably more research.

But the data so far...there might be a longer latency for this disease, and I can't say much about it, but exposure for exposure, I guess two points: mineral wool doesn't fracture along its length, so you don't have a fine..generation of a real fine fiber.

Second of all, in processes using typical mineral wool, you can use mineral wool and asbestos in the same way, the same way, and generate much less exposure with mineral wool than asbestos.

So I think that speaks for it.

Second of all, the mortality data. If the risk were as great as with asbestos, we should have picked it up, I

THE WITNESS: (cont'd.) would say, so far.

DR. DUPRE: Dr. Dement, could you just run past me again how the use of an equivalent amount of mineral wool generates less exposure than an equivalent amount of asbestos?

THE WITNESS: Okay. For example, if you take an insulation worker who is cutting a piece of insulation, one made of asbestos and one made of this manmade fiber, and during the cutting process you measure exposures to each of those, and you measure them simply by the phase contrast counting method, you will find on a number-per-number basis that the exposures for mineral wool will be considerably less.

The real reason for this, you have a fiber whose nominal or average diameter...at least in the product...is three, five, seven micrometers, and when it's...if you do get a rare fiber that becomes airborne, its airborne characteristics are such it's going to settle out very quickly and not be a source of exposure in most cases.

You have to keep in mind that that's a nominal diameter, and it has a fairly wide distribution about that, and there are some fibers that are down in the less than one micron range and are the major source of that exposure.

So it's more a characteristic of the material than it is of the operation.

DR. UFFEN: Could I pursue the question a bit about the miners?

MR. LASKIN: Sure.

DR. UFFEN: I'm interested in the gold miners and the talc miners in the studies that you participated in.

I think you can see what is in the back of my mind is, any regulatory process that establishes a limit of so many fibers per c.c. or something, which is intended for one industry, might also be applied to another industry.

DR. UFFEN: (cont'd.) We don't have a lot of asbestos mining in Ontario, but we do have a lot of other kinds of mining.

Now, if I read your papers right...and correct me if I am wrong...that in both the gold miners and the talc miners the asbestos, if present, was of the amphibole type.

That is, it was either the cummingtonite-grunerite for the gold miners, or tremolite with the talc.

Is it safe for us to conclude then that the chrysotile or serpentine type are not involved, only the amphiboles?

THE WITNESS: Yes. And the talc situation, the talc mine, there were some trace...and I'll say trace...and we did detect a few fibers of chrysotile in some of the bulk samples, but that was, again, a trace and not consistently found with all of them.

I think the Mount Sinai group...I'm thinking of Art Langer in particular...who studied this same talc mine with Dr. Messite..and looking at that material they also found trace quantities of chrysotile.

But again, it's certainly not the major source of fiber exposures in that situation. It's not one that was consistently found.

I'll say your conclusion is ninety-nine and nine-tenths percent correct.

DR. UFFEN: A regulation, then, that doesn't specify a difference, doesn't recognize a difference between the amphiboles and the serpentine, could then be applied unnecessarily against fibrous serpentines found in the tailings of other kinds of mines? Is that a fair conclusion?

THE WITNESS: We really don't have any...we really don't have any studies, epidemiologic studies, of mining populations exposed to fibrous serpentine.

THE WITNESS: (cont'd.) I think you would have to make an analogy looking at the fiber characteristics of that serpentine and compare it with industrial situations before you made just a blanket possible extrapolation.

DR. UFFEN: Including the size distribution that we have been talking about?

THE WITNESS: I think it's important.

DR. UFFEN: Thank you.

MR. LASKIN: Q. There is only one other line of questioning that I wanted to ask you about.

One of the things that we have been told by a number of people this summer that we should be doing, is considering some kind of quantitative risk assessment.

If one were going to embark on that kind of exercize, I wonder if we could enlist your assistance and comment, if you will, on some of the epidemiological studies that are in the literature, and as to whether they would be appropriate for inclusion in such an assessment, what strengths and weaknesses you see in them.

THE WITNESS: A. You want me to do that now?

Q. Can I put a few of the studies to you and ask for your comments?

A. Sure.

Q. Let's start with the one that's closest to home that we certainly heard a lot about in the published literature, and that's the McDonald miner study in Quebec.

A. I think first overall, in addition to gathering information from the published epidemiologic studies, one also needs to gather some physics information on the characteristics of that exposure. I think those in any quantitative risk assessment have to in some way be coupled together. So I think that's probably the first point of view.

A. In McDonald, there are some data that have been published. But as far as the McDonald study, the strength of it is, if you are going to look at a standard for mining, it's a mining population.

I wouldn't, by the same token, want to take, without considering the characteristics of exposure, extrapolate that necessarily for industrial situations.

Q. Any particular weaknesses to the study? For example, from a methodological point of view?

A. Well, there's a whole series of the McDonald publications. The earliest papers had obvious methodological problems, and they have acknowledged that in their subsequent publications.

I think the most recent one published about a year ago probably is a paper, if you are going to use in a risk assessment process, is a paper that you should use with those papers.

One potential problem that I can see, and it's not so much a problem in McDonald's paper as it is in the population that's being studied, presented at the Cardiff meeting was their estimates of fiber exposures for this population. Those were very high. Considerably higher than in the plant that we studied in Charleston. Probably considerably higher than many other situations.

I think you may be faced in the lung cancer case with some competing risk. For example, a person exposed to a high fiber concentration may be prone to develop respiratory morbidity and die of nonmalignant respiratory diseases, and not die of cancer.

On the other hand, lower doses of exposure may allow the person to live out a longer latency just to develop his cancer.

So I think that is at least one potential problem.

The followup methods that Dr. McDonald had used

THE WITNESS: (cont'd.) over the period of the study, the percentage followup is much, much better.

I think there is still, though, some outstanding death certificates. I'm not sure what numbers, but probably around ten percent.

I would like to see that a little more complete.

But overall, I think it's a study that is certainly worth serious consideration.

MR. LASKIN: Q. Just on your observation of competing causes of death, is that a phenomenon that in any way explains why there is only one mesothelioma in your cohort study?

THE WITNESS: A. I don't think so. I think that question in terms of dose relationships for that disease...although some of the recent data from the crocidolite gas mask workers perhaps indicates a dose gradient by time of employment...the available information on mesothelioma does indicate a strong dose dependence of the disease. It's more a function of how long you wait.

And even in my group, looking at those who are at most risk and greater than thirty years latency, there still was no...other than, again, the numbers are small...it didn't pop out as it did, for example, in the study that Dick Lemen did where he saw quite a large number of mesotheliomas in that greater-than-thirty-years latency group.

Q. I should have asked you before, are you continuing to follow that cohort?

A. The Raybestos cohort?

Q. At Charleston?

A. Yes. I mentioned the zero to six...one-to-six-months exposure group. That's still a subject of study.

Q. Do you know whether any mesotheliomas have shown up in...

A. There are not any there so far.

Q. There are no more there so far? Okay.

Coming back to other studies, can you give us any observations on Peto's work or Berry's work at Rochdale?

A. As everybody else has who has reviewed that data, I have a problem.

Q. I noticed it was absent from your comments. At least as I read through your thesis, you did mention a number of other studies but I don't believe you did mention the work at Rochdale, although I may be mistaken.

A. I don't recall. I have another paper that is coming out that does read that, but the Rochdale data I find difficult to interpret.

First of all, the estimates of exposure have changed over the last seven or eight years and it's just difficult to interpret.

Second of all, I haven't seen that it actually put together the person's work history and the estimates of exposure levels, and actually done a dose-response analysis.

I think the other problem with that when you start to break off those who were first employed after dust controls were in, the data does still indicate that there is an excess of risk, and, you know, you have the obvious question of have we sufficient latency in that group.

So from a dose-response point of view, I find those difficult to do in terms of trying to plot out a dose-response curve...at least in the published data. There may be some other data that I have just not seen, but in the published data it's very, very difficult.

Q. I take it some of the difficulties you are referring to are the changing in techniques of counting and so on?

A. They obviously had the confounding problem

A. (cont'd.) of changes in counting methods in the U.K. has been more frequent than changes in counting methods in the U.S.

Q. What was the counting method for the optical microscope at your plant in Charleston? Was it a full view or graticule count?

A. Graticule counts. Those data are presented... I've tried to gather all the data I could on what was used, and there is a table in the dissertation that tries to...first of all I tried to describe what the methods were, and also tried to get information on how they were selected.

That's presented on page eighty-one, table three two.

Q. Of your thesis?

A. Yes.

I tried to compare what was done. First of all, in most cases found varying...well, first of all, the Greenberg/ Smith impinger was used in every case except the U.S. Public Health Service study, which used the midget impinger...mainly for convenience.

The liquid was water alcohol with various proportions. Flow rates were the same. Counting cells varied somewhat in that the U.S. Public Health Service used a Dunn cell, the others used a cellugrapher.

They were all 100X bright field, and they all use...I don't have this table...but I believe all of them used a graticule type of count, not the full field.

Q. I take it that...not everybody has got a copy of this, but just for my own understanding and I suppose for all of us if we come to look at this table, in the first column, sample years collected, are those the...should we be putting a nineteen beside that? Does that mean that the data....

A. Yes.

Q. ...for example, the company's data was in 1930, 1934 and 1936 only?

A. The company insurance carrier.

Q. Yes, the company insurance carrier.

A. For the impinger was 1937/46, 1937/65, and the company is 1956 through 1971.

Q. Maybe I could just put that on the record because nobody else has it.

The insurance carrier, its sample data were done in the years 1930, 1934 and 1936. The State Board of Health in 1937 and 1946, the U.S. Public Health Service in 1937 and 1965, and the company's own program...

A. Began in 1956.

Q. ...continuously from 1956 through to 1971. So that there were no measurements between 1937 and 1946, and no measurements between 1946 and 1956.

A. I think that's correct.

Q. Okay. Just one or two studies I could ask you about. What about Dr. Weill's study, which is another study that has been put before us this summer?

A. Well, I think the biggest problem with that study is the method of followup that was used relies solely on social security information data, assuming that a person who wasn't deceased by social security records was in fact alive, and that's not valid in most cases...or in many cases.

So I think first of all he has underestimated his risk.

The data in his paper for the overall risk...the SMR's are so low that one wonders what kind of selection bias is in effect to produce such an abnormally low SMR for many causes in that cohort. I don't think I would use that data in risk assessment.

A. (cont'd.) I understand that that population is now being studied further, and I gather it should be a very important piece as well. But I don't think I would rely too heavily on that piece right now.

Q. What...if you yourself had to choose, what works would you give serious consideration to, apart from what you already told us?

A. In terms of dose-response data, I think the first thing to do is look at those studies which use surrogate of dose and years of employment as some measure of exposure, and try to place some upper bounds, if you will, on exposure levels. And look for consistency, largely.

But the papers that you have that I think...to look at dose response...are obviously the most recent McDonald paper...

Let me qualify this - dose response for lung cancer.

We have the most recent McDonald, we have the papers by Enterline, you have the Peto/Berry papers from Rochdale which shouldn't be thrown out, but need to look very closely at what they say.

Then you have the Charleston study.

There are really about four that have some attempts to look quantitatively at dose, and each one has its own limitations.

Q. Why did you put the qualification on lung cancer only?

A. Mortality is good for diseases that have a high case fatality rate. By that I mean, once a person has developed lung cancer, the probability of mortality from it is quite high.

Those diseases that don't have necessarily a

A. (cont'd.) high case fatality rate, such as asbestosis, a person may have a lot of disability but he may die of cardiovascular diseases.

Mortality is not a very sensitive technique, and so I think you have to look at respiratory morbidity studies in that context.

By that I mean, measures of lung function and x-ray changes.

Q. What weight do we put upon the work that has come out of Mount Sinai in New York, which appears to use duration of employment as a surrogate for dose, as you say?

A. That's the information I was saying that really needs a look overall at how the surrogate of dose relates to exposure. Bear in mind that different investigators, because of the cohort in hand, define the cohorts differently. Most of his are twenty or more years of employment.

So you really do have a fairly high-dose group in his insulation workers.

The other piece of information in terms of low dose is the short-term workers who they have...Herb Seidman, I guess, has followed up for a considerable period of time.

I do think that those data are worthy of looking at for short-term high, very-high dose situations, but they also extrapolate from short-term high exposures to long-term level exposure, subject to a lot of controversy.

Q. Can you elaborate on that?

A. From a toxicological point of view, if you are dealing with a substance that is metabolized and is eliminated from the body in some way, or is activated in some way to the ultimate carcinogen, then I think you really need to look closely at peak exposures.

I think for asbestos it is a material that has a

A. (cont'd.) relatively long residency time.

The question...but still the question is, given such high doses, have you overwhelmed any defence mechanisms that would be in effect for the long-term lower-dose situation.

I don't have a ready answer for that. I don't think any...there is no...the data that Hans Weill in his paper generate indicated that there was both a cumulative-dose effect in his group, and an effect of exposure level. So they are not independent considerations. They have to be jointly considered.

We really, at this point, we are stuck with the situation that even at best we don't have a population to study for mortality purposes that have had a long-term, very low-level exposure to asbestos...even in this cohort of Charleston, South Carolina.

Look at the exposure back before 1940, and even in that subsequent period. Some of those exposures were forty, fifty fibers per c.c. So you still don't have that situation.

MR. LASKIN: Thank you very much, Dr. Dement. I should turn you over to my colleagues.

I don't know whether there is a batting order, Mr. Chairman, but I gather Mr. Hardy has reserved himself to the afternoon. That's about all I can tell you.

MR. DUPRE: I guess M. Casgrain wishes to lead off.

M. CASGRAIN: Yes. I'll be very short.

CROSS-EXAMINATION BY M. CASGRAIN

Q. Dr. Dement, I was looking at tab nine and I still see that on the paper it says 'draft, subject to revision, not to be quoted'. I presume that you don't mind if I quote you from now on, but I would like to ask you, however, has this paper been reviewed in the scientific meaning of the term so that it is now a paper which can be said to have been reviewed and can be used?

Q. (cont'd.) Is that what you would say of it?

A. This paper has been reviewed ad nauseum.

Q. Within your milieu is there not some kind of procedure which is used through which...

A. Within the Institute, obviously. Within NIOSH there was a review process for this paper, and it was subject to peer review, outside review, before presentation at the Cardiff meeting.

It was also the subject of a Ph.D. dissertation, so it was subject to considerable scrutiny by that process.

Q. I don't doubt it was scrutinized. What I am trying to get at, and you might correct me if I'm wrong, I thought that one could understand that in your scientific world there was some kind of a process whereby one puts in that a paper had been reviewed and it was then classified as matter that could be dealt with.

Is that not a process which is normally done?

A. Mmm-hmm.

Q. Has your paper gone through that specific process?

A. The paper has gone through a review. More than one review.

DR. MUSTARD: I guess specifically, has it been reviewed by the journal in which it is going to be published?

THE WITNESS: It was the subject of a critique, of a discussion by scientific peers, at the time of the presentation in Cardiff.

DR. UFFEN: Is your paper in press now?

THE WITNESS: Yes. In press insofar as the Cardiff meeting is concerned.

M. CASGRAIN: Q. I would like to turn to the use of the SMR and the explanation that you gave for using the tables.

Q. You said that you preferred not to use the county in which the plant was located, and I think you said...you say so in your paper anyway...that it was because of the possibility that a number of workers in that county would have been exposed to working in a shipyard, and therefore to crocidolite...or something different than chrysotile? Correct?

A. At least your comparison population is certainly not an unexposed population, from that point of view.

Q. What I have difficulty with is, having stated this and having used another standard mortality rate, do you make any allowance in your study for the fact that some of the workers in your own study obviously would have had to be exposed to shipyard environment.

A. Yes. I have looked at the...the allowance would have to be on your observed mortality, deaths. I have looked at the ten cases that we have information on....

Q. Before we come to this, my question is the following: On your overall conclusion in your study, what I'm trying to get at is the following - you start off by saying I'm not going to use the SMR for the county because I assume that it is high precisely because a large number of the workers would have been exposed to shipyard, and therefore crocidolite...or something different than chrysotile.

Having said this, what I'm trying to get at, it seems to me to be logic I should see in your paper somewhere when you conclude that in respect of your expected and so on, that allowance has to be made for the fact that even amongst the workers that you have been studying a proportion thereof...not a specific number, a proportion thereof...would have been exposed to shipyard.

A. I don't doubt that there were some people who had shipyard employment, but to come back and adjust expected deaths is just not scientifically valid.

A. (cont'd.) You just don't have data upon which to adjust your expected deaths. It doesn't exist.

Q. I'm going to try again. Perhaps I don't understand you.

It seems to me in logic that if you were to make an assumption at the outset that you are not going to use the SMR because it's not reliable inasmuch as a large proportion of the workers of that county would have been exposed to something other than chrysotile - namely crocidolite - wouldn't it be just as normal to expect that in your own study you would say caution has to be exercised as to what I actually saw by way of death because some of those may have been exposed to crocidolite?

A. Let me answer that by presuming some facts, first.

First of all, the choice of any standard population for calculation of death rates is a very difficult one in most situations.

You have to remember that even in the U.S. population the workers who were at high risk of cancer also contribute to the U.S. mortality, so you should probably adjust down your expected deaths because some of those worked in coke ovens. So you are underestimating the true effects of the occupation.

Second of all, to choose a county population as a standard population where available data indicate that there is an effect of a shipyard employment on that population - in addition to the effect of the plant that you are in yourself - is not justified.

Thirdly, perfectness of this standard population is attested to by the fact that when I look at my one-to-six month exposure group, my very low-exposure group, I get five lung cancers observed, four expected - based on my population - which is almost exactly what you expect.

This attests for the fact that my standard

A. (cont'd.) population is not abnormally...I guess I'm saying is more perfect than imperfect.

Q. I don't want to argue with you. It's just... what I'm trying to get at and what you just said to me is that... but when you were in effect trying to distinguish between workers exposed to chrysotile, as opposed to workers exposed to crocidolite, this is where it becomes important that those allowances be made, is it not? Because this study, from what I can see here, is one in which you find...is indeed is interesting because it deals only with people who would have dealt only in chrysotile.

A. Mmm-hmm.

Q. What I'm trying to say to you is that, with due respect to you, I doubt that a little bit when in effect I see that overall your population seems to have been exposed to crocidolite at work, and I say I've got to find some of those workers who were exposed to crocidolite in that particular study, or is that something which doesn't make sense?

A. I don't think that the evidence available supports your conclusion there is a strong shipyard crocidolite effect in this group.

Where are all the mesotheliomas?

Q. Not in this group, but let's get to that issue.

In this respect, you also stated, you gave two reasons to my learned friend to talk about, distinguish between mesothelioma in that group and elsewhere. One of them was the following: You said that out of twenty-six lung cancer deaths you found that ten of those had dockyard experience?

A. That's not true. You are reading my paper incorrectly.

Q. I'm sorry. I took your answer...perhaps I took it wrong. I said either with twenty-six people and sixteen...

A. No.

Q. I thought you had answered that none have dockyard experience among the twenty-six deaths. Am I wrong?

A. I'm saying that from the U.S. Public Health Service respiratory disease questionnaires, you can conclude that these occupational...

Q. You went so fast I missed you completely. Would you mind going slower?

A. Yes. The U.S. Public Health Service had...

Q. The U.S. Public Health Service?

A. Yes. ...information from 1964 and 1971 whereby questionnaires were administered to employees of this plant. On that questionnaire, past employment was documented.

I am saying of this twenty-six, ten were included in these two surveys...none of which mentioned any dockyard exposure.

Q. I guess I completely missed out. My calculation was simply twenty-six, ten out of twenty-six leaves sixteen, therefore sixteen had dockyard experience. That's not fact, though, is it? I shouldn't make that assumption?

A. What I'm saying is, those sixteen we had no questionnaires on.

Q. You had no questionnaires on them?

A. That's right.

Q. Is it possible, therefore, that one might assume that those sixteen, perhaps some of them had dockyard experience?

A. Assume what you like.

Q. No, no. I'm asking you.

A. I don't know.

Q. It's not what I like. I'm asking you what you like me to assume. It's your paper, not mine.

A. I'm saying that I have a sample of the

A. (cont'd.) population of lung cancers, and of that sample none were exposed to crocidolite in the dockyard.

Now, the representative...

Q. Would you say that again? You say you have...

A. I have a sample.

Q. You are talking about your seven hundred and some-odd workers?

A. No. Of the twenty-six...I have a sample, I have ten...of that sample, none were exposed.

Second of all, if the dockyard were a significant confounding factor in this study, one, I should see more mesothelioma; second, in my one-to-six-month exposure group I should certainly see more lung cancers; and third, it should sort of negate any dose-response relationship, or at least have the effect of making this level off.

Q. It could be perhaps that the exposure levels are higher than those that you were told? That could also be one reason why you would see here what you just told us?

Nevertheless, it seems to me that if I look at the SMR again and I see sixty-six point five as opposed to thirty-seven point ninety-eight, to me that's about double the figure...not quite. If I look at page ten twenty-four dash eight, and what I'm trying to ask you is the following: If I took... instead of taking your SMR of thirty-seven point ninety-eight, if you used the sixty-six point five SMR...

A. That's not SMR. That's age-adjusted death rate.

Q. Well, if you wish. Allow for my ignorance in those technical terms...but if you were to use these figures... that is sixty-six point five instead of thirty-seven point ninety-eight as adjusted...and applied that to your table of

Q. (cont'd.) expected, and so on, would you not sort of have a result which would have to be half the expected?

A. It would if you considered that an appropriate standard population, but I don't consider that to be the case.

Q. No, no. So if you and I agree, it would then cut the result by half?

A. Perhaps.

Q. Well, that's mathematics, isn't it?

A. Mmm-hmm.

Q. So, if you...

A. If you consider that to be an appropriate standard population, and I don't consider it to be the case.

Q. No, but if you made that assumption, it would cut the results down by half, would it not?

A. By the same token, if you applied that halving...or the doubling of expected deaths to my one-to-six-month group, I would have a deficit of lung cancer of fifty percent, which is highly unappropriate for most populations and you would suspect some kind of a selection bias in that case.

Q. Which is why that population to you was very suspect at the outset, because it had such a high mortality rate?

A. No. The one-to-six-month group is right on in terms of what you would expect.

DR. DUPRE: M. Casgrain, may I ask a question, please?

Dr. Dement, you have mentioned your one-to-six-month exposure group. I just wonder if you could explain that to me, because I have taken the cohort as involving workers who were employed for six months or more, and I don't see where...

THE WITNESS: No, it's not in this table. I am just saying that we are continuing to followup and this is the data that are to be published.

DR. DUPRE: Oh, and this therefore involves an employee population that is not part of the cohort of this study?

THE WITNESS: It's not part of this paper. It's the same cohort, it's the same group of works - the cohort being the one-to-six-month group, for instance, as the greater-than-six-month group.

MR. LASKIN: It's being studied now?

M. CASGRAIN: Q. That's got to be reviewed later?

THE WITNESS: A. Yes.

MR. LASKIN: He is expanding his...I'm putting words in your mouth...but you are expanding your...

THE WITNESS: I'm just saying I have more supportive information since that was announced.

DR. DUPRE: Right. But nobody among the seven hundred and sixty-eight, by definition, would fall into a one-to-six-month group?

THE WITNESS: That's right.

DR. DUPRE: I just wanted to make sure.

M. CASGRAIN: Q. If we leave that for the moment, you were asked questions about the number of days you used in a year as work days, and you said you used three hundred and sixty-five.

THE WITNESS: A. Mmm-hmm.

Q. Could you please explain to me again why you used three hundred and sixty-five?

A. It is a conservative estimate, from my point of view, as to what exposures were. The employee's medical...his employee record, had month, day and year for entry into a job, month, day and year for exit from a job. Those two dates were simply subtracted from each other to find the total number of days within that period of time, and multiplied times the average

A. (cont'd.) exposure for that job during that period of time, to come up with an exposure.

What I'm saying is, my saying that, you know, the period of time obviously included weekend periods, but I didn't take them out. I just assumed the number of days were number of days.

Q. All right. What kind of work were they doing during the weekend?

A. Same as most other people do, I suppose. I have no idea of hobby exposures. Neither does anybody else, in most mortality studies.

Q. Well, let's perhaps address us to the questions.

Have you inquired from the company records or the people there what kind of work these people were doing over the weekends? Whether they were still working in the room, or other occupations that you qualified in your study?

A. There were some...I'm sure...some overtime work, especially during the war years, for most of these people, and longer than eight-hour work.

Q. Sundays as well?

A. There is some maintenance that goes on most times.

Q. What maintenance? What would maintenance be?

A. Maintenance is cleaning of machines and dust collectors, and that type of thing.

Q. So that can we assume that over the weekend you would have a certain number of employees who would be exposed to more dust, perhaps, than during the week, because they were cleaning up the plant?

A. Well, you have to remember that the plant generally operated on a seven-day-a-week basis, rotating-shift type of thing.

Q. Do you have this evidence that the plant was operating seven days a week?

A. Discussions of people, you know, in terms of records, it has been as in most plants...

Q. From 1930 on?

A. No. As in most plants, it varied. My exposure estimates, those people who were assigned to those jobs, I tried to account for the process of cleaning.

If you look in the job category that I had, I had people who were in two categories - handling of waste or unspun fiber, and cleaning. And in most cases those were quite high exposures.

Q. My question to you is, you talk about the war. The war lasted for six years at the most, so if you say that they were operating on a seven-day-a-week shift, that would be during the war. That would account for six years out of over thirty, would it not? And I'm talking about your overall period that you cover.

If I go back to the period...

A. The plant...

Q. ...if you would let me finish my question...if I go back to your period, I'm asking the question again: Excluding the war years when the plant would be operating seven days a week without stopping, I'm asking you whether in fact, checking the company records, you did not find that apart from the war years on weekends those employees working there were in fact working at cleaning the plant, and maintenance?

A. That's not a typical process. They usually are assigned to a job, and...

Q. That's not...

A. ...maintenance including his...

Yes, I have discussed all of this with the plant

A. (cont'd.) employees. They don't even have records to indicate the number of days worked. It's all from discussions and recollection of long-term plant employees who happened to be there during that period of time.

Q. So there was no record for this particular...

A. There is no real record of number of hours worked for each employee. The only record is his job, and when he worked in that job.

But by assuming a seven-day work week, I'm sure I've taken into account those situations.

M. CASGRAIN: I have no other questions.

DR. DUPRE: Thank you, M. Casgrain.

Mr. McNamee?

MR. McNAMEE: Yes, I have two or three questions.

CROSS-EXAMINATION BY MR. McNAMEE

Q. When Dr. Allison McDonald was here, she indicated that she had hired some technicians to do these conversion tables, based on probably the same data that you analyzed.

Have you had a peek at what her conversions worked out to? Yours are what, three-to-one for textile and eight-to-one for the other operations?

A. No, I haven't been given those data.

Q. You don't know whether hers confirm or question your conversion figures?

A. I haven't seen them.

Q. That shipyard that M. Casgrain was talking about, did that cease operation at the end of the war?

A. No. That shipyard is still in operation.

Q. So it has been an ongoing thing, what, since before the war, even?

A. Yes, I think before the war. It has been pretty much a continuous operation. It's now actually for the nuclear ships, one of the nuclear processing shipyards.

Q. Would it be possible, and...it would be impossible for us to segregate out the effect of household exposure. You might have a worker in the textile plant, maybe his brother or his father is working in the shipyard plant contemporaneously, is that correct?

A. That's entirely possible.

Q. Was there a large population of people still employed in that shipyard?

A. Now?

Q. Yes.

A. I don't know what the population is now. That's been fairly cyclic. I think a peak of, say, twenty-nine hundred, twenty-nine thousand.

Q. Most of the fiber counts or samples were taken by the company in your study, is that correct?

A. Mmm-hmm.

Q. Did you regard them as reliable in the sense that you didn't consider that they were either deliberately lowered, or...

A. No, no. I guess there were two reasons for regarding the data as being reliable. First, in terms of fiber counting there has always been a fairly good relationship in terms of sharing and duplicate counting of filters between the company and the U.S. Public Health Service. So there has actually been exchanges of counting and counting of filters, and that's been a good process.

Second of all, the data that was generated, in comparison with independent data collected by the U.S. Public Health Service pretty much agreed. In fact, in the 1965

A. (cont'd.) assessment values, Public Health Service, it was considerably lower, in fact, than the company data.

So I don't feel that there is any undercounting or underestimation by the company sampling program.

Q. Looking at your statistics in tables with respect to the black male workers, it would appear that if you convert these years of employment into fiber years in the same way that McDonald has categorized by fiber years, that maybe your results would indicate that in, say the zero-to-a-hundred fiber years, that probably the SMR might be very low, as suggested by some of McDonald's?

A. I haven't chosen to publish yet the black male data, for a couple of reasons. One, despite the efforts to try to followup this cohort, the followup in terms of known vital status and known available death certificates is not that good.

So it's...has quite a potential for underestimation.

Second of all, the group that I studied is very, very small in comparison.

But, I will say when you stratify the black male data by both latency and duration of employment, you find that there is at least in numbers an elevated SMR fairly consistent with the white male data, considering that the black males had a high turnover too.

So the other problem with the black male data if you look at the smoking, the questionnaire data on cigarette smoking for black males indicates that in comparison with the U.S. adult population, black males in this group were very, very light smokers. So I think you have to temper those results with that knowledge.

MR. McNAMEE: Thank you, Dr. Dement. Those are my questions.

DR. DUPRE: Mr. Starkman?

MR. STARKMAN: No, I have no questions.

DR. DUPRE: Miss Jolley?

MISS JOLLEY: I have a few.

CROSS-EXAMINATION BY MISS JOLLEY

Q. Getting back to the assumption of the seven days, I think you stated quite clearly that that would also overestimate dose and therefore all it would do is if you took, say, that they only worked five days, you would be shifting the dose down and the slope would be higher in fact, so that I think that that's appreciated.

A. I made the assumption, really, because I knew all the controversy that would ensue if I assumed a five-day work week. I knew that the question of underestimation of dose would be a major issue at that point.

Q. The other thing you did state is that you did give your exposure models to the company for comment.

A. That's right.

Q. I mean, presumably the models that appear in your study were approved by the company, is that correct?

A. I don't think approved is the word to use. I would say we both agreed from the outset that I would develop what is in essence the first three chapters of this dissertation. That would be a subject of technical review for accuracy of what was done, and any comments I had on procedures for estimation of dose. Those comments were considered and incorporated back in the final version of the paper.

Q. In the disstertation.

The other question that I have deals with something that the Commission is interested in, and that is you mentioned the fact that few people in fact die of asbestosis, that most die of other causes. In your study in particular did

Q. (cont'd.) you come up with a number of deaths from other causes that would in fact be related to asbestosis?

A. The comment that there are few deaths from asbestosis, the real point I was trying to make is that compared to mortality from asbestosis, morbidity and disability is perhaps a bigger question.

Q. Right.

A. And this group did collect whatever pathology information existed on the cases in almost everybody who died. I did not use that information to correct, if you will, the death certificates. The death certificates were taken and coded by nosologists as they were coded by physicians. There were other cases of asbestosis mentioned as either one or two things - not as underlying causes of death, but as contributory causes of death, or identified by autopsy.

In most cases in the U.S., autopsies are performed after the death certificate is filled out, so that very seldom is that cycled back to correct the death certificate.

So yes, there were some other cases.

Q. Our concern is for compensation purposes as well, as you can understand.

My last question deals with the OSHA standard, and that is something that our government hasn't included in their proposal for an asbestos standard, and that was - you mentioned in the talc, for instance, talc containing asbestos had to have an OSHA warning lable on it, and what do the warning labels indicate under the...

A. That was a recommendation that talc containing asbestos have a warning label. It has been thoroughly voluntary. Some companies have put it on, others have not.

Q. But asbestos products generally have warning

Q. (cont'd.) labels in the U.S. That is a requirement under the asbestos standard, is it not?

A. This gets into a difficult situation whereby it's an OSHA requirement. The mines and mills who produce it fall under a different government agency - MSHA - and there is no requirement to label the bags as they leave the facility.

So it really has been more up to the companies.

No, not with talc. I'm talking about talc producers.

Q. But asbestos coming into production facilities in the U.S....

A. As asbestos, certainly.

Q. ...has asbestos warnings on it.

A. Yes. But the talc situation is one that sort of fell in between the two agencies, and I don't know that it has been resolved at all yet.

The other problem is the controversy, the company in particular, really hasn't acknowledged that that material is there. So without them acknowledging it and labelling it, it's really all voluntary.

Q. I think I was almost more...

A. Let me add that other companies have..when they have found tremolite in their talc, it's labelled.

There is some inconsistency and it probably puts them at a bad market advantage, but it's done.

Q. All I wanted to actually say, though, that asbestos has to be labelled if it's pure asbestos going into a production facility...

A. That's right.

Q. ...and that's a requirement not required in our standard proposal.

A. Our standards, if you want to...for those

A. (cont'd.) materials that contain this material that is a fiber by fiber definition for counting, it's an elongated particle, and if you look at it by electron microscopy as best you can with available methodology, would say has a composition identical to asbestos, still the question is do you require a label on it. And so far, we haven't addressed that.

MISS JOLLEY: Thank you very much, Dr. Dement.

DR. DUPRE: Thank you Miss Jolley.

May I then propose that we break for lunch until two o'clock?

MR. LASKIN: I'm just wondering whether it's...

MR. HARDY: Quarter to, just to be safe for everyone concerned.

MR. LASKIN: Yes.

DR. DUPRE: Fine.

THE INQUIRY RECESSED

- - - - - - - - - - - -

THE INQUIRY RESUMED

DR. DUPRE: Well, Mr. Hardy, do you wish to proceed?

MR. HARDY: Certainly.

DR. DUPRE: Please, counsel.

MR. LASKIN: Can I just for everybody's benefit, and I'm sorry, Tim - just before Tim proceeds - clarify one matter for the record?

If we could go to tab nine, table seven. Some of you may recall that when Mr. Peto was here he questioned the figure for expected under lung cancer in the most heavily exposed, being point zero four, and I gathered from speaking to Dr. Dement at the recess that in fact that is an incorrect figure, as is the figure beside it.

EXAMINATION BY MR. LASKIN

Q. Dr. Dement, am I correct that the figure for expected should be point one three?

A. Point one three.

Q. And the SMR is one five five three, is that correct?

A. I better make sure.

Yes, that's correct.

MR. LASKIN: Has everybody got the right line?

M. CASGRAIN: Oh, yes.

MISS JOLLEY: Can you repeat it?

MR. LASKIN: Zero point zero four, under lung cancer expected for the most heavily exposed, is now point one three. The SMR, instead of five thousand, is one thousand, five hundred and fifty-three.

THE WITNESS: That correction is part of the paper for the Cardiff meeting. It will appear correctly in the proceedings.

MR. LASKIN: Sorry.

DR. DUPRE: Thank you, counsel.

Proceed, Mr. Hardy.

CROSS-EXAMINATION BY MR. HARDY

Q. Dr. Dement, from your CV which was passed out this morning, do I gather correctly that the type of work you are doing in your present job is different from the type of work you have done in the past, and that you are no longer doing studies of other peoples' plants, but rather trying to run your own?

A. I have a combination of trying to run an inhouse health and safety program in addition to doing studies both within IHS and some collaborative work continuing with NIOSH. And also an adjunct appointment at UNC School of Public Health.

A. (cont'd.) I am doing some studies with those groups.

Q. Do I gather correctly that you got into the business of looking at asbestos first by being trained as an industrial hygienist?

A. No. I got into the business by first being trained as an engineer and coming to NIOSH in 1971, and being assigned to the ongoing asbestos study for that time.

Q. At that time did you learn, I gather from your resumee, a good deal about measurement of asbestos in those industrial hygiene concerns?

A. Initially that, as far as asbestos, my primary assignment at that time was industrial hygiene assessments of asbestos processing plants.

Q. Was that in the era when NIOSH was working at perfecting the membrane filter method or improving it, at least?

A. I sort of came on the tailend of the work done previously in the mid-sixties by Eyre and Lynch, and their final publication of that method, I think, came in or about 1970 or 1971. So it was sort of the time that the data were certainly still being analyzed and looked at, but the samples had already been collected...in terms of evaluation of the method.

Q. So that by 1971 when you started in NIOSH, the basic methodology for properly using membrane filter sampling was set?

A. It had been published, yes. It had been published by Eyre and Lynch...at least the method that was subsequently recommended by NIOSH in its series of publications after that, and also the criteria document.

Q. I gather from your description that that's a method which they had been working throughout the late-sixties

Q. (cont'd.) to document and clarify and set forth guidelines?

A. Well, the method was really in terms of the Public Health Service asbestos study, which was ongoing before I came. But their protocols, in effect, essentially call for the same sampling and counting method for those studies as subsequently was published and adopted by the U.S. Public Health Service. So there was a number of years where this method was really used by the Public Health Service as a standard method.

Q. Do you know about when the method had settled down to be a standard method?

A. It really came roughly after 1964/1965 when they were looking at what index of exposure would be settled on. Some early work looked at counting fibers longer than ten microns, for example. An earlier paper looked at the relationships between various methods for sampling asbestos.

After the 1964/1965 period is when the method was really agreed upon for the study that NIOSH...or before that, BOSH...was to get underway.

Q. When you started at NIOSH in 1971, were you involved at that time in a study measuring asbestos levels in various industrial plants?

A. Yes.

Q. Did that study, by chance, include the Charleston plant?

A. The Charleston plant, I think the last industrial hygiene study by itself conducted at the Charleston plant was before I came to NIOSH. I think that was around about the early part of...the latter part of 1970, the early part of 1971. That study was conducted actually by Dick Lemen. I think you heard from him earlier.

Q. Then were the results from Charleston compiled

Q. (cont'd.) along with results from other plants? In any sort of public way, do you recall?

A. I don't remember any compilation of those data separately. The NIOSH criteria document published in 1970 did contain some summary data from the overall U.S. Public Health Service study, but I don't remember specifically Charleston in that publication.

Q. Maybe I can help you if I give you portions of two documents. I have for at least one of them the complete... I guess we have both the complete documents, if you want to look at them, but I have just taken out the relevant pages, and I've got extra copies...or at least I've got some extra copies.

Perhaps for the record I should describe the documents and ask Dr. Dement if he recognizes them. The first, which has a cover that says Occupational Exposure to Asbestos, I believe that's the 1971 or 1972 NIOSH asbestos criteria document?

A. That's correct.

Q. The second document, entitled Feasibility of Industrial Compliance with New Asbestos Standards, which has three names on it, including your own and Richard Lemen's, do you recognize that document?

A. This is an internal document that was never published. In fact, the data that you have in the criteria document are really the same data.

Q. I think it is the same data, and you shall see the pages I have attached which are from each of those documents basically are the same data. There is a little bit of difference in the data, so you may want to look at one or the other later.

DR. DUPRE: Mr. Hardy, may I ask my learned counsel if he wishes to assign a number to these documents?

MR. LASKIN: I think we had better. Let's call

MR. LASKIN: (cont'd.) them forty-four and forty-five in the order in which Mr. Hardy put them in.

DR. DUPRE: Which one being forty-four and which one being forty-five?

MR. HARDY: Forty-four would be the NIOSH criteria document, the document with the picture on the cover.

DR. DUPRE: Right. Okay. Forty-five is...

MR. HARDY: Forty-five being the document with Dr. Dement's name on the cover.

DR. DUPRE: Thank you. Excuse me for interrupting.

EXHIBIT # 44: The abovementioned document was then produced and marked.

EXHIBIT # 45: The abovementioned document was then produced and marked.

MR. HARDY: Q. Could you perhaps tell us something about the measurements contained in these documents, Dr. Dement, how they were collected, what your involvement was in collecting them?

THE WITNESS: These...the U.S. Public Health Service began its study of roughly thirty-nine asbestos plants, as I said, back in the mid-sixties - actually planned some time before that. Throughout the years, samples were collected on a rather routine basis in industrial hygiene studies conducted within each of the thirty-nine plants. Some plants were sampled more than others, and in most cases it was personal breathing zone membrane filter samples.

The data that you see here are just summaries of information from those industrial hygiene studies.

Q. Do you recall...let's turn on exhibit forty-four to the last page, which is table twenty-six...which indicates in the title that it is data from asbestos textile plants, and then

Q. (cont'd.) lists data from plants A through K... with some omissions, actually, in the letters.

Do you recall, Dr. Dement, whether the Charleston plant is represented on this chart?

A. I'm sure it probably is. I can't tell you which plant it is. The 19...I will say that rather than the exposure estimates in my study, the data from this study were included in arriving at exposure estimates. But I can't tell you which one of these are...without our key...which one is what.

Q. Let me get back to that. Let me switch topics and talk about something which you mentioned right at the end of your testimony this morning, which was a response to a question from Mr. Laskin about which studies you would review to do risk assessment for asbestos regulation purposes.

Would you also include in the studies that you look at the recent Newhouse and Berry study of the friction materials plant in Great Britain?

A. Insofar as the dose-response information contained in it, sure.

I might also add that I was speaking of risk assessment for lung cancer.

Q. Right.

A. There are other papers on risk assessment for other forms of asbestos disease.

Q. But for lung cancer you would look at the Newhouse and Berry friction materials study?

A. I think you have to look at all the data that exist. I mean, to exclude a piece of data offhand without looking at it very closely no way to go.

Q. On page one of your Charleston study, which is, I guess, tab nine, you indicate in the introduction in the middle of the page that it is well established that occupational

Q. (cont'd.) exposure to asbestos is associated with increased risk of many diseases, including...if we drop down a little...cancer of the gastrointestinal tract.

Isn't it a fact that most of the reviews of the asbestos literature and most of the experts believe that the evidence is equivocal in gastrointestinal cancer, rather than being well-established?

A. You probably find opinions on both sides. I, for one, think the data are probably a bit stronger than stating that they are equivocal. I will say that the data do vary between studies.

Q. Which might lead one to conclude that perhaps it wasn't well-established, would it not, that there is such a risk?

A. I think there is a reasonable consistency among the data to come up with this statement.

Q. In your study, if we look at table three, I gather you did not find a significant increase of gastrointestinal cancer in the Charleston workers?

A. There was no significant increase overall. The population is small.

If you turn to the dose-response data, however...

Q. Which would be table seven?

A. Table seven.

Q. Again, there are no statistically significant increases in any type category, right?

A. There are not statistically significant excesses. However, there is a trend of increasing SMR of dose. The numbers are small. I wouldn't assign too much significance to numbers.

Q. But on the basis of your study at least, there is that well-established evidence that increased gastrointestinal

Q. (cont'd.) risk is...

A. My study is too small to conclude anything about GI cancer as far as significance. I would say my data are not inconsistent with it.

Q. I would like to ask you a little about a comment you make in tab ten, which is an article which you wrote with Dick Lemen and Joe Wagoner, entitled Epidemiology of Asbestos-Related Diseases.

Could you maybe just briefly describe what this article is supposed to accomplish?

A. This is simply a review article submitted to a conference which was held. This Environmental Health Perspectives is actually, as you see, a workshop conference, and it is simply a review article. It's meant to be a review article.

Q. Meant to review the existing literature and...

A. At that time.

Q. That's what I thought, and I was particularly curious about a paragraph on page eight of that article, actually the last paragraph of the article, where you describe the results of a document often referred to as the estimates paper, the Califanno estimates paper, cited in footnote 108 in your document, about which there has been a great deal of publicity over the years.

I wondered why, in the context of a review article, you didn't also indicate that this document had also been widely criticized and its conclusions widely challenged by a number of scientists who looked at it?

A. I don't necessarily subscribe to what the Califanno article says. It's simply put in to point out that there are currently a large number of people, past employees of asbestos-processing facilities, who are now at risk of asbestos-related diseases. It's simply put there for that purpose.

A. (cont'd.) There are a lot of...almost every paper in the review has been widely reviewed and perhaps widely criticized at some point in time. It's not our intention to place in the literature necessarily those views.

Q. Then I guess...my question was sort of why, given the importance of it being most of the conclusion, and given that there had been published critiques of the document challenging it very severely, I wondered why those criticisms weren't also included in your review of all of the prevailing literature?

A. I think first of all, remember that anybody who wants to critique an article can usually find fault. These estimates are very, very difficult to generate and they are always going to be subject to considerable controversy.

I think that they are putting it here simply to make the point of the fact of workers at excess risk, not necessarily to say that these are the precise figures.

Q. Although the figures are included?

A. The figures are here. They are as presented by the publication.

I guess we put a disclaimer, 'although widely disputed'.

Q. There was some discussion this morning of the South Dakota gold mine study, on which you worked. I believe you referred to the McDonald study in the same mine as a survivor study, is that correct?

A. At least in terms of having achieved twenty-one or more years of employment. That's correct.

Q. But there was no requirement that the McDonald cohort live to any given age, was there?

A. Not in terms of age, but by having to work twenty-one years still makes it a survivor population in that likely even looking at the pneumoconiosis risk, they have a

A. (cont'd.) significant risk of pneumoconiosis - even pneumoconiosis mortality, before twenty-one years.

All I'm saying is that to define the cohort in that way you aren't necessarily sure that you have the entire picture of the mortality expressed for that group of workers.

Q. On the other hand, isn't it also true that if you didn't follow up a group of workers twenty years beyond initial exposure, which this study does by requiring that they work for twenty-one years, wouldn't there be a significant chance that you might not have a long enough followup period to find lung cancer?

A. Don't confuse followup period with years of employment. It is true that you must follow a cohort of workers twenty or more years. It doesn't necessarily mean that they had to have been employed by that company for twenty or more years.

Q. Isn't it also true that having a cohort of workers who worked at least twenty years, you probably had a cohort which was quite heavily exposed and therefore most likely, if there is an increased risk, to demonstrate such a risk?

A. It depends. I'm not sure. Because in the NIOSH study a lot of the deaths from lung cancer actually were in people who had not achieved twenty-one years of employment, but had the required latency.

But defining the group to be twenty-one or more years employed, you probably have also those in that group who have been heavily exposed to silica on at least a large competing risk for nonmalignant respiratory disease. You can't die of two causes...at least we don't code them that way.

Q. But a lot of the workers survived, in fact the greater percentage of the workers in this cohort were still alive after twenty years, weren't they?

A. And there was a large excess of pneumoconiosis mortality.

Q. But not lung cancer?

A. But not lung cancer.

Q. So you have a cohort which was heavily exposed, which was followed for more than twenty years, which had no lung cancer excess?

A. You also had a large competing risk, you had a selected population, and you also had at least a good number in this population who had never worked underground.

Q. As I understand your further discussion of that mine this morning, the NIOSH has commissioned another group to do...I think you referred to a more complete, more comprehensive study of those workers?

A. That's right - one that would be a larger population defined from plant personnel records.

Q. I assume that...let me strike that. Who is doing that study?

A. The study was contracted out to Stanford Research Institute to do at least abstract records and do followup on them.

Q. Is it fair to say that that study, because it will be more complete and more comprehensive, may be able to put to rest the dispute between the study you worked on and the McDonald study?

A. Subject to, you know, peer review of that data, it should be at least possible from that data to have a more complete group to draw more conclusions on.

Q. As I understand it, there is a draft of that study which has been widely circulated. Have you seen that draft?

A. It's a draft that is still under review.

Q. The June, 1981 draft?

A. That's right.

Q. From the Stanford Research Institute...it's SRI it's called now.

Do you know what the conclusions of that draft are in terms of lung cancer risk?

A. The conclusions...and I have to qualify this as we aren't sure the cohort is complete at this point...that is still being verified. These are not NIOSH conclusions at this point.

Q. No, but do you know what SRI's conclusions are?

A. They found no excess in lung cancer overall. When you looked at it by duration of employment, there was some trends in lung cancer by duration of employment.

I don't know if that's...it's been a while since I looked at that draft.

MR. LASKIN: Tim, were you able to make that paper available to the Commission?

MR. HARDY: Sure, I can make that paper available as an exhibit in the record. I believe, John, you've got a copy. I mailed it to you a month or so ago.

So you can make copies of it, but in addition you can make a copy of my copy.

DR. DUPRE: If you have now made it available, I think we have an available number - like forty-six.

MR. LASKIN: Forty-six.

DR. DUPRE: Thank you.

EXHIBIT # 46: The abovementioned document was then produced and marked.

DR. UFFEN: Does it have an author name that we might...?

MR. HARDY: Certainly. It has two authors.

Let me read what exhibit forty-six is. It is a document, which is labelled draft on the cover, the title of which is Miners Exposed to Amphibole Mineral - a Retrospective Cohort Mortality Study. The authors are Samuel D. Kaplan, K A P L A N, and William R. Gaffey, G A F F E Y, Ph.D., SRI International.

I'll be glad to make this copy available to you if you can't find the other copy.

MR. HARDY: Q. So at least at this point it appears that the SRI investigators have failed to identify, at least in their view from their records, an excess risk of lung cancer?

THE WITNESS: A. At least based on the draft report which you have, and based on two problems as I see it.

One is the cohort...there is some question as to whether or not all records were abstracted from the plant.

Second of all, the standard population which they used for calculation of expected deaths.

It is still the subject of critique.

Q. So I guess we can summarize it, the status is they didn't find an increase in lung cancer - you, and perhaps other persons, have some remaining questions and I think it's fair to say that this document, exhibit forty-six, is a draft.

A. That's correct.

Q. Which may or may not be changed, based on these questions?

A. I don't know if it's fair to say that these are SRI's final conclusions. You have to talk to SRI about that.

I mean, you have your draft report. You don't have their final report.

Q. Right.

Just to clarify the record, on page thirty-five

Q. (cont'd.) of this report, which I gather you have read, Dr. Dement, the authors at this point do state:

"None of the findings of the present study indicate the risk of lung cancer is related to underground employment at this mine".

And since it's going to be in the record, I guess everybody will have an opportunity to review the entire document.

I would like to talk a little bit about Charleston and the plant there, with you, Dr. Dement.

You indicated this morning that crocidolite was, to some small extent, used at this plant. Can you tell us when and where, and whether you are able to isolate those workers who were exposed to crocidolite?

A. Okay. I can read you what I have to say about it.

Q. You are reading from your dissertation?

A. Mmm-hmm..

"Chrysotile is the only asbestos that's ever received at the plant as a raw fiber. A small amount of crocidolite yarn is woven into a tape and made into braided backing materials, beginning in the 1950's. Crocidolite was never carded, spun or twisted, and the quantity ever used was extremely small - less than two thousand pounds - and all crocidolite weaving was done wet."

Two comments: First of all, at the beginning of the 1950's, following through 1975, there is really no way that exposure to crocidolite could have had a significant effect on lung cancer mortality in this group in that almost all of our lung cancer mortalities were thirty-or-more years latency.

Second of all, it's confined to one small plant

A. (cont'd.) operation done wet, and two thousand pounds is a very small quantity.

Third of all, one mesothelioma death in a plant... if they had processed crocidolite...would be a surprising find.

Q. Do you have any sort of feel for how many workers during that period in the 1950's would have been exposed to crocidolite?

A. Very few. This was a special product, talking with plant personnel, requested by one of their users, and it wasn't done continuously. In fact, it was done on one loom perhaps by just a small group of personnel. So there were a relatively small number ever exposed to crocidolite, and none in other plant operations, besides those.

I have looked back at the cases, the twenty-six lung cancer cases, and there was no association between employment in weaving during that time and the excess of lung cancer mortality, compared with the rest of the lung cancer cases.

Q. Let me be sure I understand what you just said. You said that there were no more than expected lung cancers from that group of weavers?

A. No. What I'm saying is, if you look at the lung cancer cases you don't find an abnormal distribution of those who only worked in that weaving process during the 1950's.

Q. But on the other hand, what you must be implicitly saying is that some of the lung cancer victims might have been exposed to crocidolite because they worked in the right area of the plant during the right period?

A. There were a few that worked in the area.

Q. Right, you can't rule it out?

A. No, I'm saying it's not associated...the excesses aren't associated with that one operation.

Q. Do you have any idea when you are going to

Q. (cont'd.) make available your information on the short-termers at the plant - the less-than-six-month employees?

A. That will be submitted for publication and I'm just in the process of finalizing the draft of the paper, so...

Q. It's out of your hands pretty soon?

A. It's out of my hands reasonably soon.

Q. How big is that cohort?

A. I didn't bring that information with me in hand, but the cohort is pretty large. I think there's seven sixty-eight in this group...

Q. Right.

A. ...and in addition to that, it raises it up to over eleven hundred.

So it's a sizable group of workers.

Q. The short-termers plus the existing cohort will be eleven hundred?

A. Roughly eleven hundred, yes.

Q. So we are talking about another four hundred, three hundred and forty...

A. Four to five hundred people. I think it's probably closer to five hundred.

Q. These again are white males?

A. Mmm-hmmm.

Q. I believe you indicated this morning that you do know the results in terms of lung cancer in that cohort?

A. Preliminarily, yes.

Q. It's five observed and four expected?

A. In that...yes, in that ballpark.

Q. Which, I would guess, is not a statistically significant difference?

A. No.

Q. Would it also be fair to say, or to guess, on

Q. (cont'd.) my behalf at least, that all of these workers would be in your lowest subcohort of exposure - less than ten thousand fiber days?

A. They would find themselves in there. In fact, in this analysis there will be a lower exposure subcohort of less than a thousand fiber days. So I think they are probably zero to a thousand, and thousand to ten, that type of analysis.

Q. This morning there was also some discussion of your black male cohort, which is not included in the Cardiff paper?

A. That's right.

Q. I believe you said that there were a couple of reasons at least why you didn't include the black male data, and one was that the cohort was quite small?

A. Well, it's more than just the size.

Q. You also said there were more short-timers?

A. Well, they are short-time employees. The followup...the real problem with presenting the data in a published form at this point is that the quality of the followup, and especially of death certificate ascertainment, is not really up to conventional standards, I would say, for epidemiologic studies. And that's despite a lot of efforts to try and follow these people up. It's considerably harder and much more time consuming effort than we had to go through for white males.

Q. But that group is not all that much smaller than your white group, is it? If we look at table five point eight, which is on page 170 of your dissertation, we discover there are a hundred and fourteen deaths in that group, which is not all that different from the hundred and ninety-six deaths in your white group?

A. But you don't have all the deaths either. There's a large, quite a large percentage of outstanding death

A. (cont'd.) certificates. So any analysis that you would undertake of that would underestimate risk significantly.

Q. Would underestimate it if in fact the people lost to view were different from the people who you didn't lose to view, right?

A. That's right.

Q. Which may or may not be true?

A. I have no way of knowing that. That's the reason we haven't published.

Q. In fact, that is guaranteed to underestimate it, right?

A. It's likely to miss a few lung cancers. I'll say that.

Q. It's also going to miss a few people who didn't die of lung cancer?

A. You have to remember that those people who were lost to observation in the calculation of expected deaths were assumed alive. So two problems...

Q. Wait a minute. You don't have to assume they are alive, do you?

A. No. I'm telling you the problems in expected deaths. There is a problem in expected deaths and there is a problem with observed deaths, and the trend for both of these data is to underestimate it.

The observed...the expected deaths were based on person-years contributed by people that we have no information on vital status on. They were assumed alive. In fact, quite a few have probably died.

So we've had too many person-years. We've overestimated expected deaths.

Conversely, we have likely underestimated the

A. (cont'd.) observed deaths by not having death certificates. So both factors combined have a combined effect of underestimating the SMR.

Q. But those underestimates presuppose that you are going to assume people are alive, and you are going to assume something about those people for whom you don't have death certificates, neither of which assumptions you need to make to nonetheless present the data. Correct?

A. That's not true. If you don't have any information on an individual, you don't have any ability to assign his death to any given date. So you have to go to either one of two things - you could stop his person-years accumulation at the point where you lost him, or you can assume he is alive.

Q. Or you could just exclude him from the study?

A. That's not a well-thought-of epidemiologic approach.

Q. Could you explain why you couldn't do that as long as you, you know, obviously...

A. You are dealing with the situation...

Q. ...disclose the fact that you weren't doing it?

A. You've got a situation of potential selection biases, and the cohort that you throw away versus the cohort that you keep. You have no information to base a decision one way or the other.

Q. But isn't the solution...isn't a possible solution to that to admit, in describing what you did, that there were a certain number of people who you would like to have in the cohort, but you don't have in the cohort, who are a certain percentage of the cohort, about whom you don't have information and who may bias it one way or the other, but you just don't know - that way avoiding going through procedures that necessarily under- or

Q. (cont'd.) overestimate risks?

A. You would have to assume one of two things, and your assumption by throwing them out is that what you have left is representative of what you threw out. There is no information to make that assessment at all.

Q. But no information one way or the other doesn't mean that you should assume that the remaining data is useless, which you do when you don't publish it?

A. I haven't assumed that it's useless. I'm saying I haven't published it yet. I'm still working on the followup.

Q. Maybe we could talk a little about the results that you did have and at least were willing to put in your dissertation. If we look at table five point eight on 170, as I look at that table I gather that the disease results among the black cohort are quite different than the disease results for the white cohort?

A. If you accept these data without qualifications, sure. You could say that.

Q. Okay. But just given the data you've got today, with all your caveats which we have heard, you've got a lower-than-expected death rate for all causes?

A. Mmm-hmm.

Q. And a similar lower-than-expected death rate for lung cancer among the black males?

A. That's correct.

Q. It is...I mean, you've got more than a hundred deaths there?

A. We only have four lung cancer deaths. I don't think any statistician would hang his hat on four lung cancer deaths.

Q. But on the other hand, if I had asked you how many lung cancer deaths you would expect in this group, based on your white data, it would be a number considerably greater than four, wouldn't it?

A. A number, if you applied white male death rates to this group. I understand what your question is, I suppose.

Q. Well, you found a relative risk for lung cancer for whites of...for the whole cohort, which is what we are looking at now...of three and a half, right?

If there were a similar relative risk of three and a half for blacks, you would have found considerably more than four lung cancers, right? You would have found more than fifteen?

A. Mmmm-hmm.

Q. And I would assume that the difference between four and fifteen is probably statistically significant?

A. But you are basing your conclusions simply on the two numbers, and there are a lot of caveats to that conclusion.

If you look at the table that presents the death rate information for this county, versus U.S. death rates which were used...

Q. Right.

A. ...you find two things: The U.S. death rates use to calculated expected deaths were higher, considerably, than the county, and considerably higher than contiguous counties by several orders of magnitude.

Second of all...

Q. Can you tell us what table you are referring to?

A. Page 178.

So you overestimate expected deaths, at least using that, based on the data that would have suggested that you have done that.

Q. So what you are telling us is the county rates for blacks for lung cancer is lower than the national rate? In the same way...

A. No, I was talking about...no...I'm looking at the state, looking at the state.

Q. Mm-m-hmmm?

A. You can't draw much of a conclusion based on looking at one county.

Q. Of the state rate for blacks?

A. It is still considerably lower...

Q. Than the U.S. rate?

A. ...than the U.S. Right. That's correct. Second of all...

Q. The county rate for whites was much higher than the national, the state rate for whites?

A. If you look at the trend in the black male death rates, the county rates in the county in which this plant is located, looking in it relative to the rest of the state, they are elevated with respect to the rest of the state - still suggesting the effect of that shipyard, perhaps, on the counting of death rates of blacks.

Second of all, look at cigarette smoking data for this population, presented on page 180, and compare it with U.S. death...with prevalence of smoking in the U.S., on page 182.

You find that compared to U.S. males, the prevalence of smoking is considerably less among black males in this population.

Q. As between 1965 and 1971?

A. No, that's for the smoking data...

Q. Which is the small sample of smoking data that you've got for the total cohorts?

A. Oh, no. That's the smoking data for the

A. (cont'd.) total cohort. I have 1964 and 1971 questionnaires, so the time frames are comparable. I don't have smoking data for everybody in the cohort. Just a sample of it.

Q. Right.

A. But it suggests, at least, that...

Q. And it's a sample based on those workers who happen to be still at the plant in 1964 and 1971, which may or may not have been representative over the whole history of the plant?

A. Who knows? It appears to be at least reasonably representative of plant operations at least.

Q. Just while we are on smoking data, this smoking data does not tell you anything about whether these workers... let me back up...does not tell you anything about when these workers started to smoke and how long they have been smoking, is that correct?

A. That's correct.

Q. Nor how heavily they smoked?

A. It's a common practice in epidemiologic studies to classify workers as current, former or never-smoked.

Q. Despite the fact that we know that how long people have been smoking and the age they began smoking, and how much they smoked, all would be relevant factors if we could know them, in determining how important smoking is in lung cancer rates?

A. That's true of almost every study. Most data you don't really have...in most studies...this is an exception... in most studies you have no idea of even a sample of the population's smoking prevalence.

So I would say compared to other epidemiologic study, I have more data. It is certainly consistent with the pattern observed for the lung cancers for this group.

I might also, when you stratify...even in the black male population...there was a high turnover, so you have quite a group in this population that have, over their lifetimes,

A. (cont'd.) very low cumulative exposures.

If you stratify this group by latency and duration of employment, and that data are at least presented in a few places here in this dissertation, you find when you do that there does appear at least in numbers...I'm trying to find the statement that is made in here...

Q. You mean talking about the four observed lung cancers?

A. No, no. I'm talking about stratification.

Okay, among black males employed more than ten years and achieving more than twenty years latency, we had three lung cancers observed versus less than one - zero point nine six expected.

Numbers are small. It's obviously not significant. It is consistent with the trend.

We also observed the excesses in other nonmalignant respiratory diseases - table five dash twelve, page 175.

Q. But the fact remains, doesn't it, that the dramatic increases found in the whites at low levels of exposure do not show up in the black males deaths? There is a discrepancy in the results there?

A. I'm not drawing a conclusion based on the data that I have in this dissertation. I think it's too preliminary to say.

Q. Do you have any idea when and if there will be further publication of the data on the black males?

A. I'm following it up, and when it gets to a point where the followup and the death certificate ascertainment is up to established norms of acceptable papers for publication, then I'll publish it. This is as much as I have right now.

Q. I would like to talk about the work you did in converting the particle counts to fiber counts, which I gather

Q. (cont'd.) was the first step that you took in your exposure determination.

If we had a graph of the paired sample data that was done by the Public Health Service in 1965, and had particle counts on one axis and fiber counts on another axis...and I think we've seen over the summer similar tables from the Canadian data in mines...do you have any idea what that sort of group of dots would look like?

A. It would have considerable scatter about a regression line, as you might expect from trying to equate two methods of measuring exposure - both of which have a high degree of inherent variability about them, and you wouldn't predict correlation coefficients to be very high simply based on that.

Q. So that the mere fact that that diagram was pretty much something like a scatter would not necessarily mean that it wasn't possible to come up with some meaningful conversion values?

A. That's right. You are really...you would predict that type of variability looking at the individual samples - paired samples.

The object of the exercise is to be able to take one group of data from the impinger and arrive at an estimated exposure based on average or some measure of central tendency to that distribution of conversions, to arrive at some...to predict some mean exposure for fibers.

The object is really not to correlate the two techniques per se, but to come to some predictor of average exposure.

So I'm not...you know, I think in the literature there has a lot been made of the fact of having those, when you plot those things out, there's a lot of scatter.

Q. I think this summer we have seen the Gibbs and LaChance scatter diagram and it has been made fun of by some witnesses.

A. Well, I'm sure it has. However, I can plot...I can take impinger samples or two membrane filter samples and plot those against each other, taken simultaneously, and these are the same methods of measurement, and I'm going to get a lot of scatter as well. Both have inherent variability in them, and it's not insignificant.

Q. Both, the membrane filter has inherent variabilities and so does the impinger method?

A. They are both counting methods.

Q. Referring again to the mines, in terms of measuring particles am I correct in my understanding that in the mines there would be a good deal of materials generated both from the mine pit and in the mills that would get counted as particles, that wouldn't be fibers?

A. It's difficult to predict what that would look like in terms of what the sampler would see. I think it's probably true that in the mining, and at least the initial stages of the milling, you have long fiber junk, just nonfibrous material. But, whether or not the impinger, for example, collects it, and even if you collected it, whether or not you counted it, it's just another debatable point.

So I think it's true, basically, that you see a lot more particles versus fiber in milling. I'm not sure that you could ever use that information to come up with conversion because of the sampling characteristics of the impinger.

Q. One of the reasons I ask is that you seemed to take some comfort this morning in the fact that McDonald's most recent conversion figure, which is in his Lyon paper, is about three, particles to fibers, which is the number which for most operations

Q. (cont'd.) you determined in your study. I think we have heard a lot of testimony this summer that higher conversion factors might well be expected in the mines, particularly compared to textile plants where there wouldn't be much other of this junk in the air, and I wanted to get your reaction to that.

A. I don't know. I think you have to weigh the generation of more nonfibrous material that would be counted as particles, versus the collection efficiency of the impinger which is not that good for fiber. You are basically counting nonfibrous material in most cases...or things that you can't really easily distinguish as fibers.

I think perhaps...I'll be surprised if the relationship didn't go the other way - have a higher conversion in the mines and mills than you would see in textiles.

Q. Could you explain why you believe that? It certainly sounds contrary to what other people have testified to this summer.

A. If you have a situation where you are generating particles and fibers, in the textile situation you are probably going to generate some longer fibers that at least have the ability to be collected and even counted at a hundred X magnification.

On the other hand, in the mine and mill - even though you might have some other particles there - the impinger is not an efficient collector of fibers. It's much more efficient as a collector of particles.

So it's likely to escape collection of a great number of these small asbestos fibers as well.

Q. Now, as I understand it, you computed your conversion data, conversion figures, on the basis of two sets of data.

A. That's right.

Q. The first set of data or paired samples collected by the Public Health Service at this plant in 1965, and as I heard you this morning you talked about there being a hundred and twenty such samples.

A. Mmm-hmm.

Q. Is it possible that there are more than a hundred and twenty pair of samples, that there might be a hundred and twenty-six and there were some that you didn't actually employ in your work?

A. The 1964 report, I think, is a hundred and twenty. I don't know. I would have to look back at the individual report to find out if there is some reason why a sample was not valid or not. I don't know at this point without looking at the raw data.

Q. And these were paired samples...

A. There were a hundred and twenty that were valid samples that were used in the analysis.

Q. These were the paired samples that were done by Lynch and Ayer as part of their project?

A. These hundred and twenty pairs, I would say, were collected by Lynch and Ayer. They were collected by the Public Health Service. I think Gerry Lynch might have written the report that went back to the company.

Q. Was there counting at that time in the fiber measurements of fibers greater than ten microns in length, and also total fibers, and also fibers greater than five microns? How were they counted?

A. The procedure that was used during that time frame was that yes, they did count greater than five and greater than ten. The report that actually went back to the plant contained only the greater-than-ten counts. Discussion with plant personnel even before this study was initiated, they were

A. (cont'd.) provided with the greater-than-five counts.

So it's a question of whether they were counted and whether or not they were reported back. This was during the time period they were obviously looking at what to count.

Q. I'm not sure I understand that. The report that went back to the company had greater-than-ten microns and total fibers?

A. That's right.

Q. Now where did you get data on the fibers-greater-than-five-microns figure conversion?

A. That's right. They were counted. They just never were reported back in his early report.

Q. The numbers exist somewhere on a piece of paper?

A. The numbers existed. The numbers were reported, the company requested these data years later, and those numbers were reported to the company.

Q. The fibers greater than five, and that's the number, the data set you used for your work?

A. That's right.

Q. Now as I understand the way you treated the paired samples from a methodologic viewpoint, is that you used the linear regression statistical technique?

A. I would say a linear model, or linear correlation, yes.

Q. You were dealing with an independent...with variables which were the membrane filter counts and the impinger counts, both of which - as I think you said earlier - had high coefficients of variation, a lot of variability?

A. That's right.

Q. I'm no expert, but I'm been told, and I'm

Q. (cont'd.) curious to get your reaction, that linear regression is not a proper statistical method to use when you've got independent variables with significant coefficients of variation. Is that an issue you worried about before you did it?

A. I certainly considered other methods. I think it depends on whose statistician you talk to.

Q. Are you your own statistician?

A. I certainly have statisticians who were on my dissertation committee. I profess to be a student of statistics.

Q. But there is evidently some...at a minimum there is some debate among statisticians about whether linear regression is the proper technique to use the way you did?

A. There is going to be debate among statisticians on how you calculate an average, but yes, sure. The method that was used for the impinger and the membrane filter paired samples was to try to at least account for some of that variability by a conversion of the data to make it more amenable for use in that process.

Q. But you just used one method which some statisticians would say is the wrong method, is that right?

You didn't try to determine the conversion by other suggested statistical methods, did you, to determine whether there were different results?

A. I tried to do one thing that Dr. Weill presented at a meeting that I attended, was to take ratios of numbers and simply calculate an average of that. I looked at the median ratio, which I feel was a better...yes, I looked at other techniques and they yield substantially the same technique (sic). Unless you calculate an average ratio, which is an average of log normal distribution, it is not a measure of central tendency.

Q. There may be other techniques too, which some statisticians would recommend...

A. I'm sure there are.

Q. ...for data with these sorts of problems?

A. I'm sure there are. There always will be, too. No matter what techniques you use, it's going to be the subject of discussion.

Q. Different techniques could give different results when you get this sort of indefinite data...

A. I would be surprised if techniques that legitimately looked at the characteristics of the two sample techniques would come up with substantially different results. There may be slight differences, sure.

Q. Let's talk about the exposure information which you then used after coming up with the conversion figures.

As I read some of the information that John Laskin went over with you this morning, particularly if we look at table three dash two in your dissertation, at page eighty-one, from the period of 1937 until 1956, there is only one set of exposure information...

A. 1937 to 1966? No, that's not correct.

Q. I said 1956. 1937 to 1956.

A. There is the 1965...well, 1937 and 1956.

Q. Right.

A. There is State Board of Health data collected in 1946.

Q. Right. That's the only data point for a period of nineteen years?

A. That's correct. Well, roughly...like, well, the Public Health Service study was, I guess, conducted in 1937 and published in 1938.

Q. And as I read table three point one on the previous page, page seventy-nine, that's four samples, four numbers, four measurements?

A. The 19...

Q. The 1946 State Board of Health.

A. That's correct.

Q. So you do have a nineteen year period in this plant with just four measurements?

A. In most operations, that's correct.

Q. What do you mean by 'in most operations'?

A. Well, this State Board of Health study was conducted at the request of the company to evaluate some controls that had been put in place subsequent to the 1938 Public Health Service study. So at least in terms of changes in plant operations there are some data, preimposed changes for that period of time.

You have to remember, controls in this plant were all in place for 1940 - basically all there as a very stable plant operation.

Q. But what we have in 1946, is that the State Board of Health came in and did four measurements?

A. Evaluating a specific operation.

Q. Right.

One thing I did notice in your description of dust technology in this plant is very much discussion of use of water or oil or other liquids to contain dust clouds.

A. Water, especially.

Q. Was there much of this done in the plant? Did it vary in terms of how much was done over the years?

A. The use of wet process is...early on there was...I'm saying the early thirties...some work in terms of adding oils prior to the...actually prior to the spinning operation

A. (cont'd.) or carding operation, I suppose, some blending...the things were called blending oils.

This was used...not long. Mainly because...well, I just have anecdotal information on the reasons why, but apparently it didn't really do as good a job from a product point of view. They had some problems with the product, so it was discontinued.

The use of wet processes in the plant in terms of actually wet processes, as opposed to ventilation controls, was fairly recent except for weaving, and weaving, I believe, and there is a table which I tried to document, after all of this discussion of what each plant operation did, and that occupies a good number of pages. But I tried to summarize major changes.

I'm trying to find that.

Q. Is that page 115, table three dash fourteen?

A. No. This is page forty, table two dash two.

You'll find that the small looms which were weaving a light-duty cloth were always wet. Between thirty-seven and seventy-five...and the detailed discussions of that are contained in the paper itself...there is a combination of local exhaust and some wetting before weaving, really.

Some other operations which, I guess there was some wet spinning which was begun in 1966, although looking at the industrial hygiene data and talking with plant personnel, that was not a universal application and was really not very effective at that point.

Q. So what you are saying is, they started using wet spinning but they didn't find an impact on the exposure levels?

A. They didn't really adopt it universally. It was more experimental than anything else, and I guess the reason for it is that it didn't appear that effective, at least as they were doing it, at the time, in reducing exposures.

This plant has subsequently to this study, in 1975,

A. (cont'd.) converted to everything done wet, from fiber receiving through weaving.

Q. That was in 1975?

A. It's been a gradual phase-in of that process.

Q. And that phase-in began before 1975?

A. It was a completely separate process in another building, and there was some experimental work that was going on prior to that time. But it has been within the last two or three years that that has been a complete conversion.

Q. I'm curious about that because I think an apparent anomaly is to me, when you look at table six, which is your exposure information in your study, is that you determined that for a number of operations in the plant for many, many years that for some things like draper weaving and universal winding, for the entire period from 1930 to 1975, that there was no change in exposure level?

A. Mmm-hmm.

Q. That strikes me as certainly very uncommon in the industry, where you find great changes over the years...

A. Draper weaving...

Q. ...and secondly, I guess, a little hard just to believe that over forty-five years there wouldn't be changes in exposure levels?

A. Well, you know, you can't simply look at the levels themselves. You have to look at the overall picture.

First of all, draper weaving was always done wet on the same looms that existed in the plant in 1975, in the same building, and there was a remarkable consistency in the data over that time period.

So without any knowledge that things have changed, I think the most appropriate assumption is that exposures were reasonably the same, at least as far as the average exposure was

A. (cont'd.) concerned.

The models that I tried to use in this process of estimating exposures tried to take into account changes in controls and processes. In this case there was no apparent change.

Q. I guess what you also must be telling me is how the equipment is run and how it is maintained and how much attention is being paid to it hasn't varied over a forty-five year period either?

A. Well, as I...

Q. Especially in an era when there is more and more recognition of the need to control asbestos exposures, which is obviously very different in 1975 than it was in 1930?

A. It may have been in 1975 that probably...you have to remember the 1938 study was the source of our five million particle per cubic foot standard which was in existence all the way through until the 1971 NIOSH publication that recommended a two fiber...eventually a two fiber standard.

This plant was in compliance with that. No reason.

Q. Also, I guess it's a little curious that if you look at carding between 1946 and 1965, and compare it to carding in the following ten year period, we find exposure averages going up, and I think you find the same pattern in ring spinning between the first two periods. That, too, strikes me as unusual and not what you would have expected.

A. The process for doing the...fitting the model was one, to account for every change of process and look at jobs. The last step in fitting the model was to look at any residual trends in the data that I had not accounted for by my other variables in the model, and these are the two operations that appear to have some residual trends in exposure.

I have some discussion of that in the dissertation, and it's presented in a factual manner.

Q. Do I gather what you did here is you had a model for exposures in the plant?

A. That's right.

Q. And that model to begin with took into some account technological changes, and then you plugged in the data based on the measurements you had to see whether in fact changes do show up in the data due to the technological changes, and if some statistically significant change in the data occurs then you took it into account? But if you couldn't find some significant change, then it wasn't taken into account in the exposure numbers you used. Is that the way it works?

A. That is roughly the way it works, but the real idea of the models is to validly predict exposures, so the statistical criteria for allowing a parameter to stay in the model was relaxed to the point one level, whereas customarily it's zero point five.

Q. So it's a slightly relaxed statistically significant test?

A. It's a relaxed statistical criteria.

Again, the idea is one of valid determinants of exposure, and not so much statistical, strict statistical validity. Validity of exposure estimates is of primary importance.

Q. Your underlying assumption must be that unless the change was of some magnitude, it was not a change that you were willing to give credence to. Is that one way of looking at what you just said?

A. That's...within some guidelines. If you look at what actually happened, in terms of statistical validity, look at the parameters for the regression model, table 315, page 117, you'll see...I have a footnote in that table that indicates those parameters which were entered into the model simply because they satisfied the zero point one level of significance, but not the

A. (cont'd.) point zero five. There are very few, very few of those.

If you look at the engineering controls, there are none of those...that when plant personnel and records indicated that situation did change, did not the statistics indicate that in fact it had been changed in a very significant way.

Q. I'm also curious in understanding the model of how you handled people who worked in each of the general areas. Maybe if we look at table six, we can set the stage and I can ask the question.

As I understand it, in each of the general areas you have some exposure numbers?

A. Mmm-hmm.

Q. Which go down the first column. And then you have subcategories which refer to particular jobs within each zone, which you display in the columns to the right, is that correct?

A. Yes.

Q. As I also gather, you have some employees who weren't assigned to any of the particular jobs on the right, but instead you know worked in the general area.

A. There were four...and this is based on some very interesting work done by Dr. Corn and Esmen, in their studies of mineral wool plant exposures, where in addition to looking at segregating the plant by area, also developed what they called uniform job groupings within a given area.

Those uniform job groupings being to group jobs within areas in such a way that they are based on knowledge of the operation, of what they did, that exposures would be reasonably the same.

This is what was used in this study. Within each of the areas, there were four major job categories: One, the general area people - and that included people who worked throughout the

A. (cont'd.) plant operation, were not specifically assigned to a machine for operation in the area, but were throughout the operation. My estimate of that person's average exposure was taken as the overall mean for all things in that area.

I had other groups called machine operators, and I had within each group sort of subgroups. Machine operators were allowed in the model the average exposure for the area plus some increment in exposure, simply because they work at the machine itself.

That increment was based on breathing zone and personal sample data, so I have an average plus an increment.

The same goes for cleanup personnel, people who were sweeping plus fiber handling.

So I have tried to account for the fact that general area sampling does do a decent job for people who were roaming through an area.

People who are operating machines experience that general area sample, general area concentration, but also some increment simply because they are closer to the machine. It is also an attempt to account for static versus personal sampling.

So I tried, at least in this model, to account for those variabilities.

Q. Now, as I understand it, if we had an operator in preparation waste recovery, which is the first category in your table, in 1930, and he's a subcategory one operator, his exposure, you would say, is seventy-eight?

A. Based on fitting my data to the models that I generate for each operation, that's what I would say his average exposure was.

Q. Okay. The question I have is, the tough one is, a worker who is in that general area in 1930. Do I gather correctly that your model takes the average of all the numbers all the way across...

A. No.

Q. ...through 1930, since he would be in the general area plus doing some of the things in that general area?

A. No.

Q. That's not what you...?

A. No. That general area sample, in fact, includes nearly every sample in that area - everybody's. It's the area average.

If you look at table 315, you'll see that I have parameters for regression model, and they go all the way across.

Take preparation - these are on a log linear model.

Q. What page is that?

A. Page 117.

In order to generate this subcategory one exposure, you would add...you would take the general area parameter and add to it parameters for the other operators and convert that from a log model to an average...a mean for a log normal distribution, basically.

So what I'm saying is, there was calculated some increment because of this person's operation, to the general area's data, and that increment was generated by the linear model.

Now, you can't add across.

Q. Okay...

A. The expression that was used to convert that number...

Q. That's the way your model is set up, so that...

A. That's right. It is set up to look at general area, look at an increment, and add it to the general area because of the operation.

Q. What about maintenance workers? How did you handle them? I heard a lot this summer that maintenance workers have incredibly extraordinary peak exposures, and there has been

Q. (cont'd.) some questioning over the summer about how you estimate their exposures. How did you do it?

A. Maintenance workers were not included in my cohort. My cohort was defined as asbestos production workers.

Q. Ah. That's news.

A. No, that's stated in the paper.

Q. It is? I missed it.

A. It says, "The cohort was limited to seven hundred and sixty-eight white males employed six or more months in textile production operations".

Q. Oh, I guess I just assumed that included...my mistake. I assumed that a maintenance worker is employed in a production operation.

A. No. The maintenance workers at this plant, they do maintenance on facilities in textiles and run over and do maintenance on the rubber operation as well.

Q. So we don't have any maintenance workers in this study?

A. The only workers that you have within the area that would be categorized as maintenance, and there's probably some debate if we could call it that...if you will look at the appendix to this thing, I have the actual exposure...

Q. 'This thing', your dissertation, right?

A. Yes.

You will find...you've got a fixer.

Q. We're on page...

A. No, you can look on any of them, basically.

Q. Okay. Page 228 has the fixer on it.

A. Right. That's the only type of person. That's a pretty standard textile classification for that operation, so those are the only people who are included in this cohort.

Those people would spend full time within the operation.

Q. A fixer is a man in foster winding who fixes the foster winding...

A. No. I think his job is one...his job is actually to go when a thread breaks, retie it and make sure it takes off again.

Q. Oh, so he's not really a maintenance worker?

A. He is maintenance insofar as that operation is concerned.

Q. But he is not maintaining the equipment?

A. No, he is not coming in and dismantling the equipment and starting all over. That's a separate department - engineering.

Q. So in other words, you consider him as a...

A. A general area person.

Q. A general area person?

A. That's right. He is all over the facility.

Q. You don't give extra exposure to him?

A. That's right.

Q. The way I guess you would expect a maintenance worker to have extra exposure when he's working...

A. But he is not a maintenance worker in that sense. He is not blowing off machinery. He is running throughout the operation.

Q. As I understand these data which you are working with, both the impinger counts and the sixty-five fiber... 1965 fiber counts, those are all area samples?

A. Which ones are you referring to?

Q. The impinger counts are all area samples, right?

A. No. The Public Health Service impinger counts were breathing zone. Many of the insurance carrier impinger counts were breathing zone. Many of the company's own industrial hygiene samples were in fact breathing zone.

Q. You call them breathing zone. Isn't there a difference between a breathing zone sample and a personal sample, as we now refer to it, membrane filter samples?

A. No, not really. A breathing zone sample is one where the person is actually followed and the sampler is held within the zone that the person would ordinarily be breathing in.

The reason for that, in 1930 we didn't have a small pump that we could hang on a person's belt, so he had to be followed physically to take that sample.

Q. I had an understanding that with the sort of midget impingers they were using, physically you just couldn't hold the thing right up wherever the guy went, with his head, isn't that right? You've got a big-sized pump and this guy is moving around working...it's not like a personal sampler that you have attached to it, is it?

A. It's a good approximation to it.

Q. Could you maybe tell us, describe it to us, give us a feel for it?

A. The impinger sampling that historically has been associated with breathing zone samples is actually one of a hand-cranked pump held on a person's belt who was sampling, and holding the sampler within the person's breathing zone and actually following that worker.

Q. Is that what was done in this plant?

A. What was done at this plant, they did have some power, so there was a cart that had the pump on it. The cart was moved with the worker.

You have to remember too that a lot of workers are fairly fixed in the textile facility. They aren't doing a lot of running around, so it's quite a bit easier to collect a breathing zone sample.

Q. I'm not sure I...you've got a cart sitting next to the worker?

A. The card is mobile. It is actually moved.

For example, a person in a ring spinning operation would actually go back and forth in this line of ring spinners and service them. The cart was actually pulled with that person.

Q. Someone else was pulling the cart back and forth?

A. That's right.

Q. And on the cart was the sample collector?

A. A pump.

No, he was holding the sample collector in the breathing zone. On the cart was simply the pump, they had to roll it around.

Q. Who was holding...

A. The person doing the sampling was holding the sampler.

Q. Okay.

A. In this breathing zone.

Q. So what we've got is a man working on a loom or on a ring spinner, moving around to some extent?

A. To some extent.

Q. And there is a man there with him who has got a cart that he is pushing, on which is a pump, and a sampler which he is holding...

A. Yes.

Q. And he is running along...or moving along, at least, back and forth...

A. That's precisely how it works.

Q. And trying to keep the collector somewhere near the breathing zone?

A. Either that or he has a hand-crank pump.

Q. Is there anything about that collector that makes it have to be held at a certain angle or it spills, or...

A. If you don't hold the impinger upright, you have a pump full of impinger. Basically that's the only criteria.

Q. There's liquid and it pours out?

A. It will go into your pump.

Q. So it seems to me there would be a difficulty in moving that...

A. I don't argue it was difficult. I'm just saying that it was done. They were called breathing zone samples. And they...breathing zone is close to static samples where you just hang it on some zone.

Q. Right. So it's an attempt to...

A. It's an attempt at getting breathing zone concentrations.

Before the coming of the battery-operated personal sampler, that was what was done not only with impingers, but with every other industrial hygiene sampling procedure.

Q. Do you know to what extent that sort of sampling does in fact give results that are the same or different from personal sampling, as we now refer to it?

There must be some differences, given the clumsiness of the effort.

A. You know, I don't know of anybody who has published a lot of data trying to compare breathing zone versus personal. There are a lot of data comparing personal versus static sampling, and most industrial hygienists sort of accept the breathing zone sample as being a good estimator of personal exposure, and that's the reason they were collected.

Q. Turning to exhibits forty-four and forty-five, which are the NIOSH data from eight textile plants, we indicated previously that the Charleston plant was included in these eight plants.

A. I would suspect that somewhere in this data is a 1971 study done by the Public Health Service in the Charleston plant. I don't...without the key to it, I don't know which one is which.

MR. HARDY: Mr. Chairman I am almost done, but if we could take a quick recess we could clear it up and I could finish very quickly.

DR. DUPRE: Our deadline, Dr. Dement, is four o'clock, was it?

THE WITNESS: I have a five...

DR. DUPRE: Your plane is at five?

Okay. Well, we certainly can afford to take ten minutes, I think, Mr. Hardy. Shall we rise for ten minutes?

THE INQUIRY RECESSED

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THE INQUIRY RESUMED

DR. DUPRE: Do you wish to proceed, Mr. Hardy?

MR. HARDY: Yes, Mr. Chairman.

MR. HARDY: Q. I might ask Dr. Dement to look at exhibit forty-four, which is the criteria document, table twelve, the third page. I might just say that during the break I tried to see if I could help Dr. Dement identify which of the plants, A through K, was the Charleston plant. We were unsuccessful at that effort, but I think we can ask a couple of relevant questions nonetheless.

On table twelve, Dr. Dement, as I read that table for the operation 'carding', in the various eight plants, textile plants, sampled by the Public Health Service in the early 1970's, the highest average readings were in a plant, plant A, and based on ten samples, were twenty-seven point three fibers per cubic centimeter. The lowest was plant G, which were six point one fibers per cubic centimeter.

Though we can't identify the Charleston plant in

Q. (cont'd.) terms of A through K, I guess we do know that it's somewhere between six point one and twenty-seven point three for carding.

A. Mmm-hmm.

Q. Is that fair?

A. Mmm-hmm.

Q. Now, if we turn to your study on table six, which has the estimated mean exposures, and look at carding, we discover that for the entire period from 1936 through 1975, for the general area 'carding', your exposure estimates are numbers lower than even the lowest plant found in the nation in this Public Health Service study.

In other words, the lowest plant found of the eight was six point one, there were others going all the way up to twenty-seven point three in the Public Health Service study, but your numbers are five point three, two point four and four point three, going all the way back forty years in carding in this plant.

A. Mmm-hmm.

Q. I guess...it appears to me that at least for carding there is something unusually low about your exposure numbers.

A. I guess a couple of points. First of all, the data that you have indicated, table twelve, are the results of a single survey at a single point in time. Those data were in fact used...the data from the plant in question...was in fact used in deriving the exposure estimates in my study.

You will note that those data talk about carding and lump all the jobs in carding into one number. If you look at my table six, I have exposures that range all the way from twenty-two point eight down to two point four, so I'm well within the range of the data in this table.

Q. Although the bulk of your numbers, in your general area numbers, are all well below even six point one...

Q. (cont'd.) which is the lowest number among eight textile plants?

A. At a given point in time.

Q. Is it fair to say that the numbers that the Public Health Service found in the Charleston plant in 1971 were higher numbers than the numbers you used in your model for exposure?

A. I think...

Q. They were higher than six point one, we know that. Right?

A. I think in order to evaluate the exposure model you have to do a bit more than look at one point in time.

Look at table...page 125, table three seven, in the dissertation. This was my attempt to evaluate these exposure models. Given that I've calculated the parameters of the models, what if I compare breathing zone samples by operation and by job, I can evaluate how well this model predicts that average.

Q. Right.

A. I think it's unfair to take one point in time, 1971 and one survey. There are equally more data which are below that exposure level.

If you look at the Public Health Service 1965 study, it's significantly lower than the company's own sample data. So, you know, how do you relate the two? I think you have to look at the whole picture and use the data in that regard.

Table three dash seven gives my ninety-five percent confidence interval from my model an average or mean exposures calculated from the U.S. Public Health Service 1937 study, and again using 1970 through 1975 personal sampling data.

Q. That's the company data?

A. No, that's everybody's data, and included in that is 1971 U.S. Public Health Service data. It's every piece of data that exists for that plant for that period of time.

A. (cont'd.) If you will see, my exposure estimates are generally well within the confidence intervals of the...my confidence intervals generally will bracket those average exposures calculated from these two data sets.

Q. Perhaps you could explain to me, while we are looking at table three dash seventeen and then we'll get back to the 1971 samples in a minute, but on table three dash seventeen, in the column for personal sample data you have some places where you give the range, and other places where you give the single number.

I guess I don't understand why there are ranges sometimes.

A. Okay. Mainly the ranges are given when there's some possibility of two different types of job. For example, in cop winding, although we called it one job category, when you look at the data there were people who were doing slightly different types of cop winding. So I simply put it in there to indicate...

Q. That indicates really two different jobs, it's not a range?

A. Well, it's a range...

Q. So all you do is mention two different jobs?

A. Yes, it's a range for that job classification, given that there's, you know, some minor differences in terminology within that sector.

Q. See, the 1971 Public Health survey at Charleston, as I understand it, had about two hundred samples? So it was a fairly extensive industrial hygiene survey of that plant at that time?

A. Well, it was...these data were collected in basically the same manner as would be done by the company's own sampling program. So, you know, why weight that data any more in terms of relevance than the bulk of data.

A. (cont'd.) You are taking the two hundred and twenty samples and saying well, they are higher, when in fact, between 1970 and 1975 there were many, many more samples collected...probably in the neighborhood of two thousand samples.

That, to me, is a better judgement of overall exposure.

Q. Let me just make sure I understand that answer. I guess the first thing you are saying is that in fact, as was indicated by looking at exhibit forty-four, the 1971 Public Health Service numbers are in general higher numbers than were found by the company in its sampling or...

A. I'm saying that these data don't stratify any of the information that's stated by job. Perhaps if you look over the whole operation, average things, yes. But you have to look at the job, and this data doesn't present any information by job.

For fiber preparation, my table two twenty-six lists eighteen jobs.

Q. I just thought I heard you say as part of the previous answer that yes, the 1971 Public Health Service numbers did come out higher than the company numbers in subsequent years. Is that true or false?

A. Based on just averaging by carding, perhaps. They also indicate that it was consistently...there are also numbers that are lower than my estimates as well.

As you would expect on any point estimate of exposure, some of them will be higher and some of them will be lower. It's a point estimate.

Q. And so...then I gather that when you did this comparison in table three dash seventeen to see whether your model jived with some of the existing data, you just folded the

Q. (cont'd.) Public Health Service data in 1971 in with the company data to come up with that third column of numbers?

A. That's all the data collected in that plant. I would say roughly two thousand samples went into the calculating of these numbers.

There is an attempt to do a little statistical comparison on page 127, and simply breaking up exposures by ranges and seeing whether or not the models would predict exposure levels within the same ranges as the averages, and of thirty-four comparisons, all but three were well within the range and...three?... yes...and there's no indication of any bias in the direction that the averages went.

Q. Let me just make sure I understand one thing. This table we are looking at, table three dash seventeen, I just wonder if it's a figure of comparison. As I understand it, your model was based on the data from the plant and the predicted numbers, and now what we are doing is seeing whether those predicted numbers from the data agree with the data.

I mean, aren't we going around in a circle with this table?

A. A statistician would, of course, like to have a completely independent source of data to make comparisons. In a real world situation where you try to use all the data, no.

As far as statistical independence, they are statistically independent.

However, the data that was used to calculate these averages is only a small portion of the data used to generate the model estimates.

So in essence...

Q. Wait a minute. We were just talking a minute ago about two thousand personal samples. That's a pretty...

A. But still, that's about a third of the overall data.

Q. It sounds like a pretty substantial amount of the data to me.

A. It's still a third of the data.

You don't have independent data source to verify your model with. I wish I did.

MR. HARDY: I have no further questions, Mr. Chairman.

DR. DUPRE: Dr. Uffen, any questions?

DR. UFFEN: I have one. I think in answer it will probably be one minute, just so that I understand the importance of the latency period in the dose-response curve.

You used fifteen years and when I looked at the figure...if I can find it here...figure two, where you've got the SMR's for lung cancer?

THE WITNESS: Yes.

DR. UFFEN: Again, this is tab nine, I'm using tab nine.

If you had used ten years as the latency period... I'm not suggesting any reason why you would...but if you had used ten years, would the slope of your dose-response curve be lower or greater?

THE WITNESS: If you used ten years as the beginning point for person-years, you would probably get some rough estimate of that, in fact, by looking at table four.

You would have added overall, and if you used between ten...just say between ten and nineteen years, and not fifteen, you have added about one more expected death overall, and no observed deaths.

It would have a minor effect, but not...

DR. UFFEN: You would have added to the accumulated

DR. UFFEN: (cont'd.) dose five years, the way you did it when you were...

THE WITNESS: Well, add it to the dose? No.

A person's dose began to accumulate when he came into employment. His person-years would actually begin...I began them at fifteen years.

If you moved back and say well, why don't you begin at ten...

DR. UFFEN: Do I misunderstand this statement then? It's on page five, and it says:

> "For this reason, the present dose-response analyses were restricted to those achieving fifteen or more years since initial employment. This was accomplished by beginning the accumulationg of person-years for each worker after the fifteen year latency period was satisfied."

THE WITNESS: Yes, that's right.

A person's cumulative exposure would begin to be calculated from first employment. But you would not add his person-years until he achieved fifteen years latency.

That's because the first fifteen years in most cases is not considered at risk for occupational respiratory cancer, so that's the reason for that.

If you move back, like I say, it would add at most one expected death to the overall expected, and no observeds. It's all observed for greater than fifteen years.

It would be a minor contribution. I don't think it would significantly affect the duration of the line.

DR. UFFEN: Thank you.

DR. DUPRE: Dr. Mustard?

DR. MUSTARD: I have two questions. One of them goes back to the shipyard workers, and I think you stated that in a case of ten of the lung cancer deaths you had Public Health surveys which indicated whether the workers had or had not worked for the shipyards, and none of them had...that is a sample out of the total group that had cancer.

Since they did that survey, did they not survey other workers in that plant, as well as those who had cancer, or who developed cancer?

THE WITNESS: Yes. There was obviously some survey of everybody in the plant.

DR. MUSTARD: What proportion of that larger population had worked in the shipyard?

THE WITNESS: I don't have that in proportion. There are a few people who had mentioned some periods of shipyard work, none of whom were lung cancer cases.

DR. MUSTARD: I guess what I'm...

THE WITNESS: It's a small proportion.

DR. MUSTARD: ...I wanted to have some feel for what the movement is like between those two operations in that area at that time. That survey would...the total survey would give one some feel for that, wouldn't it?

THE WITNESS: It would give at least in two points in time a number or a portion who had past employment in shipyards, but it's most important for those who were cases, and that's the reason for concentrating on the cases.

DR. MUSTARD: I can see that. I would just be most interested to know what the total picture is. Is it possible to get that information?

THE WITNESS: Sure.

DR. MUSTARD: I wonder if we could make arrangements to secure it for the record?

MR. LASKIN: Sure.

DR. MUSTARD: My reason for asking that, that is one of the major criticisms that keeps being brought up and I think the additional information would be helpful.

The second question goes back to the other major problem that as a commissioner I have listened to, and this is the question of the crocidolite exposure, which is small and was during the 1950's. The early-1950's or the late-1950's, do you know?

THE WITNESS: I think the operation began in the early-fifties, as I remember, and was discontinued...it was very sporadic and I don't know exactly when it was discontinued.

But a total, according to the plant engineering personnel, a total of about two thousand pounds of crocidolite yarn were ever processed in the plant.

DR. MUSTARD: It would be possible, therefore, from the record to be able to collect your study population so that you could look at those people employed in the plant in the period after they had stopped using it, is that possible?

THE WITNESS: Well, if you did that you wouldn't have sufficient latency to observe any lung cancers because you have a maximum latency of about fifteen years. So you wouldn't have enough information to do an analysis.

DR. MUSTARD: You would have, let's see, since 1955 and you went up to 1980, you would have to...

THE WITNESS: No, you would have to continue it. My cutoff, followup date is 1975. You would have to...

DR. MUSTARD: So someone could do that, to sort of get at that question, because it seems to me that this is one of the studies which really does significantly, as you have identified in your articles, raise the question about chrysotile in manufacturing processes as a health hazard. But there still is the argument, if I can put it to you, and then I would like to hear you shoot it down, that if in effect the action of asbestos

DR. MUSTARD: (cont'd.) fibers is when you get your first exposures of it in a high enough dose, and the latency really sets the stuff in process, then maybe a lot of the after exposure isn't as important as that early exposure. We could get into an important argument about what that two thousand pounds would really mean to your work force, if one took that particular kind of stand, and I would like to hear you shoot that full of holes.

THE WITNESS: The problem is, you have a small number of people who are involved, so you really don't have a cohort, really, to do any work on. And the second problem is just a lack of sufficient latency period to even begin, in this study, to start having a significant effect on mortality.

I'm not saying that at some point in future time you may not be able to find two mesothelioma cases among people who wove crocidolite yarn, but it has not had an effect on this population as yet.

DR. MUSTARD: Have you had any way of identifying which possible groups of workers might be affected by it? Are the records good enough for that?

THE WITNESS: Only in so much as saying, looking at textile weavers. That would be the only real information except perhaps, depending on recall, who had worked in operations, the plant personnel.

DR. MUSTARD: Okay, so your basic...

THE WITNESS: The total number who ever worked there is very small, according to people I have talked with. You know, in the neighborhood of a few to ten people or so.

DR. MUSTARD: Thank you.

DR. DUPRE: Just a few questions, Dr. Dement. I understand from something you said earlier that you are separately examining a cohort of very short-time employees, one to six months?

THE WITNESS: (no audible response)

DR. DUPRE: Is it unfair to ask you if you see anything there? I mean, are there any indications of excess mortality?

THE WITNESS: I'll say that the data in the one-to-six-month group, there's no...there was a number and it's five versus four. It's not significant in the excess for lung cancer.

There are no other real differences in terms of...there are, in fact, I think, four...I can't recall the numbers...but there were a few cases of nonmalignant respiratory disease mortality there. I don't think those are attributed to asbestosis.

DR. DUPRE: If we go to your main cohort, the seven hundred and sixty-eight, the minimum entry point is six months of employment.

In your tab nine paper, the category you have, if I remember right, is less than ten years of employment, so that would be less than ten years and more than six months.

Did you ever internally break that down into some of the component parts - like less than two years and more six months? And did that show anything in terms of mortality?

THE WITNESS: It shows...we have the same sort of trends in terms of lower with less employment and greater with more employment. It doesn't really alter the trend.

In using duration of employment, you are also using sort of a surrogate of a person's dose. Really, you are substituting that for his cumulative dose, and so it is a sort of...but the trend is important to biological plausibility.

I don't think there was any dramatic peak and then drop off in exposure, in the SMR.

DR. DUPRE: Would this lead you to be wary of

DR. DUPRE: (cont'd.) arbitrary compensation guidelines that require ten or more years employment for an industrial disease deemed due to occupational exposure?

THE WITNESS: I think in compensation the issue needs also to look at, in addition to years, at what the circumstances were for that person's employment - what kind of ...

DR. DUPRE: It would mean that you might perhaps want to look at the plant in full?

THE WITNESS: It needs a look at the level, in addition to the period.

DR. DUPRE: Are you suggesting then, Dr. Dement, that in a plant where dust conditions perhaps could be shown to be especially bad, compensation guidelines might perhaps be adjusted accordingly to try and take account of the circumstances of that...?

THE WITNESS: Compensation is a tough issue. I don't want to evade the question, but the problem of compensation is one of individual susceptibility to a disease and it is very hard...it's easy for us as epidemiologists to look at a group of people, but to take that data and go right down to individuals, say well, my dose-response data indicated that at your level of exposure you are not supposed to develop asbestosis - but he has it.

It's the individual's susceptibility to disease, and I think it has to be the individual, not so much that type of grouping.

DR. DUPRE: As for my other area of questioning very quickly, if you would indulge me in a bit of administrative history for a moment, the two fiber standard in the U.S. was brought in in 1972, is that correct?

THE WITNESS: The two fiber standard really arose out of...NIOSH had made a two fiber recommendation. There were public hearings on the OSHA proposed standard of two.

THE WITNESS: (cont'd.) At the public hearings it was decided that a five fiber standard would be put into effect at that time, to be lowered to two, I think in July of 1976...is that the right place...so it was lowered to two automatically in July of 1976.

DR. DUPRE: So basically from 1972 on, you were on a descending standard from five to two, which was...and two was to be accomplished by 1976...

THE WITNESS: By 1976.

DR. DUPRE: ...article five in that, 1972.

Had there been a standard prior to 1972?

THE WITNESS: There had been a standard based on the impinger, I think it's five million particles per cubic foot.

DR. DUPRE: Five million particles, yes.

THE WITNESS: There were a couple of guidelines by the American Conference of Governmental Industrial Hygienists, who originally started with the five million particles and eventually changed that to twelve fibers per c.c., and they have sort of a history of coming down too. There were guidelines, and I guess the government enforced that five million particle standard based on the contracts...what did you call them...what's the Act called?

MR. HARDY: The Davis...the Walsh-Healy...

THE WITNESS: The Walsh-Healy Contracts Act, of government contractors, basically.

So that was somewhat the history of that.

DR. DUPRE: Now, if one just takes the arbitrary three-to-one ratio, five million particles would be roughly fifteen fibers. I asked that question because when I saw exhibits forty-four and forty-five, I couldn't help but ask myself as a professor of the public administration by vocation, the extent to which there may well have been a number of plants in 1972 who were having one

DR. DUPRE: (cont'd.) hell of a time meeting the five million particle standard, let alone the two fibers you were trying to get them down to.

THE WITNESS: At the same time, when you look at the highest versus the lowest you see that at least...if you want to accept that as feasibility for meeting the standard...the feasibility, to me, did exist.

There's a lot of voluntary compliance with the five million particle guideline, and this company, to me, is one that really tried to follow the guideline.

DR. DUPRE: Your Charleston plant, you believe, was essentially always within whatever guidelines were recommended?

THE WITNESS: They really were striving to stay within the government's guidelines, within their processing.

DR. DUPRE: Any further questions, counsel?

MR. LASKIN: No, Mr. Chairman.

DR. DUPRE: Well, Dr. Dement, may I indeed thank you very, very much indeed for your visit here with us.

I assume, counsel, that we are now adjourned sine die, or are we adjourning until November 13th, when Dr. Gibbs returns?

MR. LASKIN: We are adjourning until November 13th.

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THE INQUIRY ADJOURNED

THE FOREGOING WAS PREPARED
FROM THE TAPED RECORDINGS
OF THE INQUIRY PROCEEDINGS

Edwina Macht

EDWINA MACHT